TRANSACTIONS

OF THE

THIRTY-SEVENTH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

HELD AT

MONTREAL, CANADA

SEPTEMBER 19-23, 1932

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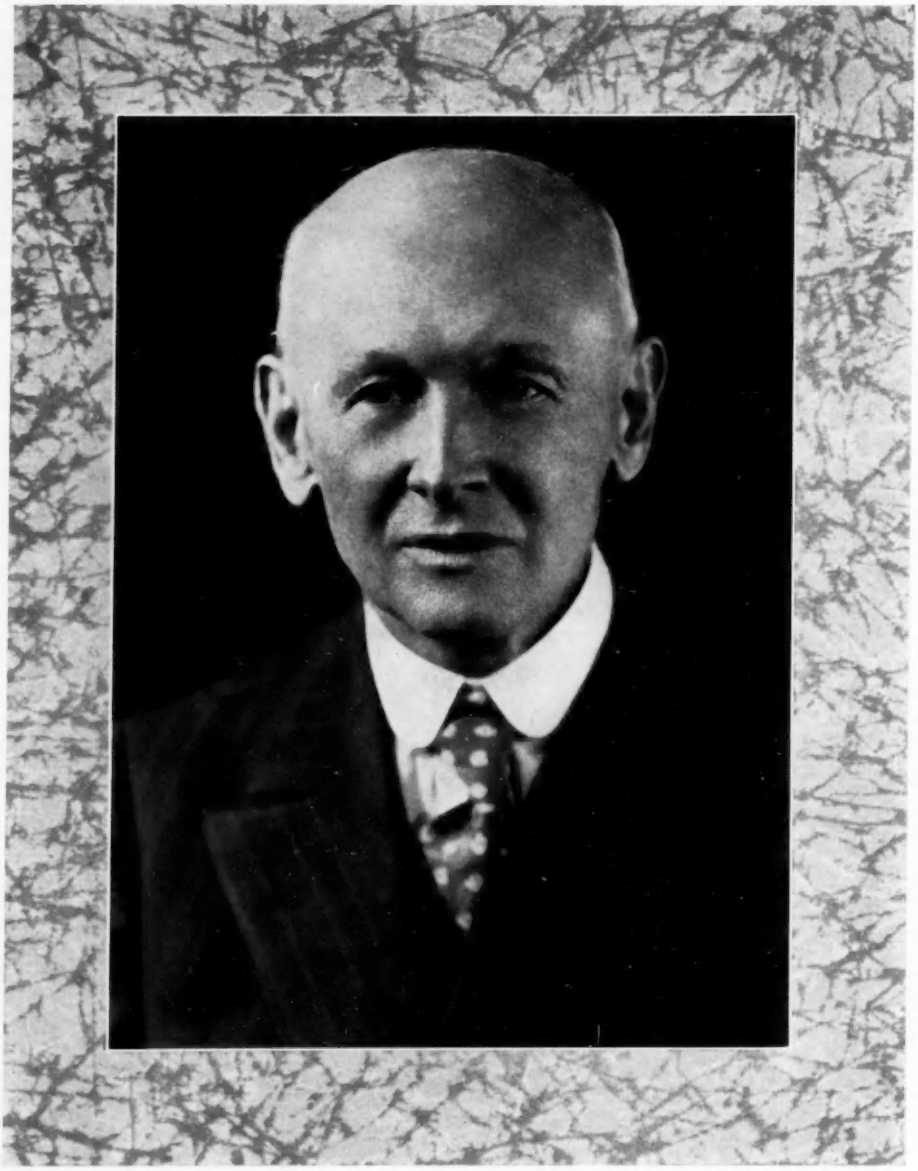

HAL FOSTER

A.B., M.D., F.A.C.S.

Dr. Hal Foster was born at Fosters, Alabama, July 7, 1858. He received the degree of Bachelor of Arts from the University of Alabama in 1880, and Doctor of Medicine from the University Medical College of New York City, now University and Bellevue College, in 1882.

Postgraduate study was taken at the New York and the Manhattan Eye, Ear, Nose and Throat Hospitals; subsequent work was done in London and other centers.

Dr. Foster has been most active in elevating the standards of otolaryngology and has unselfishly given himself to the betterment of his chosen specialty, especially in Kansas City. There he organized the Throat, Nose and Ear Clinic at St. Margaret's Hospital. For years he was head of the Ear, Nose and Throat Department at the Kansas State University. He was an instructor at the old University Medical College of Kansas City, is now visiting otolaryngologist to St. Joseph's, Trinity and St. Margaret's Hospitals. He was a charter member, is an ex-president and is still active in the Kansas City Academy of Medicine. He was the second secretary of the Jackson County Medical Society. He is a member of the Missouri State Medical Society, American Medical Association, a Fellow of the American College of Surgeons, charter member and ex-president of the Kansas City Society of Ophthalmology and Otolaryngology. He is an honorary member of several otological societies and has always been much interested in organized medicine. He was the first vice president of the Selective Service Medical Advisory Board, No. 2, during the World War, and is a member of the William T. Fitzsimmons Post No. 8, American Legion, Kansas City.

Dr. Foster was one of the founders of the American Academy of Ophthalmology and Otolaryngology, served as its secretary during the first three years of its existence, was first vice president in 1920, and was made a life member in 1921. He has missed only three of the annual meetings.

The Academy, desiring to honor one of its founders and its first secretary, affectionately dedicates this volume of the Transactions to him.

A. Howard. McKee

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1932	Dr. S. Hanford McKee Montreal, Canada	Dr. Gordon B. New Rochester, Minn.	Dr. S. H. Large Cleveland, Ohio	Dr. William P. Wherry Omaha, Neb.	Montreal

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JOINT SESSION

PRESIDENT'S ADDRESS

S. HANFORD MCKEE, M.D.

MONTREAL, CANADA

"A WISE NATION preserves its records, gathers up its muniments, decorates the tombs of the illustrious dead, repairs its great structures, and fosters national pride and love of country by perpetual references to the sacrifices and glories of the past." With these thoughts in mind I have looked through some of the early records of our Society.

In January, 1896, Dr. Hal Foster of Kansas City, Missouri, sent 100 letters to men in the western, middle western, and southern parts of the United States inviting them to meet in Kansas City on April 9th for the purpose of organizing a special Society. As a result of these letters about thirty representative men of these sections met at the Midland Hotel in Kansas City. The meeting was called to order by Dr. J. H. Thompson, president of the Kansas City Academy of Medicine, and an address of welcome was delivered by Dr. C. Lester Hall, president of the Missouri State Medical Society. Adolf Alt was elected chairman, and Hal Foster, secretary. The program for the first meeting was a long one, there being twenty-nine papers. On the day following, April 10th, a regular business meeting was held and the Society was organized. Adolf Alt was elected president, W. C. Pipino, first vice president; W. L. Dayton, treasurer, and Hal Foster, secretary. After a great deal of discussion, the Society was christened "The Western Ophthalmological, Otological, Laryngological and Rhinological Association."

The second meeting of the Society was held at the Planter's Hotel at St. Louis, where the standard requirement for members was raised from one to three years of special practice before becoming eligible for membership. At this meeting there were seventy-five papers on the official program; however, only twenty-eight were read and discussed.

The third annual meeting was held in Chicago, April 7th and 8th, 1898, under the presidency of B. E. Fryer. At this meeting, Dr. Hermann Knapp gave the Society an extended talk on the use of antiseptics, especially in operations for cataract extraction. It was decided at this meeting to publish the transactions of the Society.

The fourth meeting of the Society was held at New Orleans under very unfavorable weather conditions. The Society had as the guest of honor, George T. Stevens of New York, who addressed the meeting on "Historical Notes Relating to Anomalies of the Eye Muscles."

During the fifth year of the Society, its prospects did not seem so bright; dues were coming in slowly, expenses were increasing, former staunch members were becoming lukewarm, so it was decided to invite men from the West and Middle West to joint the Society. W. L. Ballenger of Chicago became secretary and made a very successful drive for membership. During his two year term 125 new members joined the Society.

At the seventh annual meeting held in Chicago in 1902, a committee consisting of Casey Wood, Adolf Alt, B. E. Fryer, Edward Jackson, Harold Gifford, and H. V. Wurdemann was appointed to revise the constitution and by-laws, and at the meeting in Indianapolis in 1903, this committee made its report. This, with a few minor changes, was adopted and "The American Academy of Ophthalmology and Otolaryngology" was brought into existence.

The meeting held in Chicago in 1905 was a large one and the growth of the Society was testified to by the admission at this time of ninety-three new members. At this meeting the Academy for the first time met in two sections. President Loeb acted as chairman of the Ear, Nose and Throat section, with J. J. Kyle as secretary, while D. T. Vail, first vice president, acted as chairman of the Eye section, with G. T. Suker as secretary.

At the eleventh annual meeting in 1906, the following amendment was adopted: "That there shall be a Council composed of the president, who shall be chairman; two ex-presidents next preceding him, and four fellows of the Academy, two to be chosen each year and to serve for a term of two years." At this meeting it was decided to publish the Academy's proceedings and in the secretary's report at the next annual meeting, the Academy

was congratulated upon the publication of its transactions. At this meeting Secord Large began his service as treasurer.

The fifteenth annual meeting under Wendell Reber at Cincinnati seems to have been a very successful one. Sidney Stephenson of London was the guest of honor. President Reber in his address, among other recommendations, made one which provided for the establishment of a fund "to be awarded annually or bi-annually to any member of the Academy who shall have made some single contribution to ophthalmology or otolaryngology."

In 1912 the Academy held its first meeting outside the boundaries of the United States at Niagara Falls, Ontario. Elschnig was the guest of honor and spoke upon sympathetic ophthalmia. Mr. Heath of London was also present at this meeting and spoke of his work on the mastoid.

At the eighteenth annual meeting at Chattanooga, President John W. Murphy, in his presidential address, said: "I also wish to congratulate the Academy upon its most excellent financial condition. We have $3,424.00 in the treasury and all bills paid to date. This has been accomplished in the face of the fact that a large proportion of the dues are given back to the members in the form of the substantially bound volume of the transactions and the indexes of ophthalmic literature and otolaryngology compiled by members of the Academy, and the further fact that our annual dues are much less than those of most other societies. It simply speaks volumes for the business-like method of your worthy treasurer, Dr. Large."

Walter Parker in his address in 1922 said, "Last year was a memorable one in the annals of the Academy, owing to its entrance into the field of education, which marked a distinct epoch in its history." The results of the work of the section of instruction and pathology have been most gratifying and being so well known to you all need no further comment from me.

The present position of the Academy at this our thirty-seventh annual meeting is as follows:

Our membership is about 2,025. During the last year we have spent as follows: Research, $4,500.00; Trachoma, $250.00; Physiological Optics, $250.00; Army Medical Museum, Washtington, $500.00. Our total assets are in the neighborhood of $100,000.00. It may well be said of the Academy, "Achievement

is but another milestone along the pathway of progress — the end of the journey lies ever beyond."

The most casual observer will see from these figures that our growth has been really phenomenal and, in considering Academy affairs during the last few years, I have often wondered whether we are not moving forward a little too quickly, and whether the time has not now arrived when we might justly take stock of our surroundings, rest a little in our uplifting and consolidate our position before again moving forward, taking time to turn our minds toward the less material side of our professional work.

The first code of medical ethics that we know of for the government of medical practice was that of Hammurabi, dated 2250 B. C. The monument on which it is recorded was discovered in 1901 on the Acropolis of Susa. Hammurabi was the sixth king of the first dynasty of Babylon, and the obverse of the monument shows the king received the laws from the Sun God.

This code and the Smith papyrus found in Egypt give us our earliest knowledge of conditions in the medical profession. We learn that even in the earliest times physicians' charges were controlled; carelessness in practice was punished by law, and warnings were issued against poor judgment and rash surgical intervention.

A second landmark, set up about 1,800 years later, is to be found in the work of Hippocrates, the great Greek physician of Cos. These Hippocratic writings, as we might have expected of one who was a contemporary of Pericles, Socrates, and Plato, reveal a high conception of professional responsibility, noble moral ideals and lofty aspirations for considerable medical behavior.

It is significant that the Greeks did not bother about details of "ethical practice." The guiding principle was that the physician should help, or at least do no harm to suffering humanity. To be certain of good character on the part of physicians it is probable that the several groups of teachers at the Asclepieia and Iatreia (the health temples and public medical centers) required an indenture of persons training for the practice of medicine. This probably developed into the famous oath of Hippocrates. Its chief provisions were that the physician should call a consultant if in doubt; be reasonable in fees, and if necessary or

possible forego them entirely; lead a pure and moral life, trying to be, in the highest sense of the word a philosopher, and respect and honor his teachers. The physician should not give nor sanction the giving of a poison, cause nor encourage abortion, use his position to debauch a patient nor any member of the patient's household, divulge information about a patient, advertise in any manner, nor assume ostentation in dress or manner. These simple criteria of professional dignity were the ideals of medical etiquette for over two thousand years and are still the traditional basis of modern codes. Practically every great leader in medicine has echoed the admirable ethical principles of the Hippocratic writings.

At the beginning of the last century a publication appeared which deserves to rank as a prominent landmark of progress in these matters. Percival's "Code of Medical Ethics," a classical book in its best sense, is so good that it has served as a paradigm for nearly all books and pamphlets that have been published since that time. Thomas Percival was born September 29, 1740, in Warrington, Lanc. His parents died when he was three years old, and he grew up under the guidance of an elder sister. Handicapped physically by poor vision and headaches, Percival was a scholarly and judicious thinker, highly respected for his personal charm and character. He was greatly interested in social problems and his whole life was influenced by love for his fellowmen. With the rise of industrial manufacturing, Percival became impressed with the necessity for improving factory hygiene, and advocated regulations for ventilation, rest rooms, and general cleanliness. He was not only an excellent private practitioner, but he also engaged in philosophical and experimental inquiries. The topics of encouragement and consolation which he addresses to the sick derived efficacy from a countenance expressive of a benignant sympathy and genuine goodness of heart, and an elegant and impressive character from the abounding stores of an elegant and cultivated mind.

In the code proper, Percival emphasized the combination of "tenderness with steadiness" and "condescension with authority" in the management of hospital and charity cases. Discussion of the case before the patient, particularly when the outlook is bad, should be avoided. Secrecy when required is strictly to be observed, and females are to be treated with the most scrupulous

delicacy. Talent for the profession consists in personal qualifications, which are not to be transferred: experience, discrimination, presence of mind, fertility of resources, exquisite tact, above all law, capable of deciding in the critical hour, and a discretionary power like equity, having no established rules. Percival's discussion of conduct in private and general practice is fully as interesting as his comments on hospital practice. The same moral rules of conduct prescribed towards hospital patients holds good. In dealing with the physician in later life, Percival says, "The commencement of that period of senescence, when it becomes incumbent on a physician to decline the offices of his profession, it is not easy to ascertain and the decision on so nice a point must be left to the moral discretion of the individual. But in the ordinary course of nature, the bodily and mental vigor must be expected to decay progressively, though perhaps slowly, after the meridian of life is past. As age advances, therefore, a physician should from time to time, scrutinize impartially the state of his faculties that he may determine *bona fide* the precise degree in which he is qualified to execute the active and multifarious offices of his profession."

"The physician is one whose relation to life and health are of the most intimate character. It is fitting not merely that he should possess a knowledge of diseases and their remedy, but also that he should be one who may safely be trusted to apply those remedies. Character is as important a qualification as knowledge, and if the legislator may properly require a definite course of instruction, or a certain examination as to learning, it may with equal propriety prescribe what evidence of good character shall be furnished. These propositions have been often affirmed."—*(Mr. Justice Brewer, United States Supreme Court).*

"I hold every man," says Lord Bacon, in the preface of his "Elements of the Common Laws of England," "a debtor to his profession, from the which as men of course do seek to receive countenance and profit, so ought they of duty to endeavor themselves by way of amends, to be a help and ornament thereunto. This is performed, in some degree, by the honest and liberal practice of a profession; when men shall carry a respect not to descent into any course that is corrupt and unworthy thereof; and preserve themselves free from the abuses wherewith the same profession is noted to be infected. But much more is this

performed, if a man be able to visit and strengthen the roots and foundation of the science itself, thereby not only gracing it in reputation and dignity, but also amplifying it in profession and substance."

For the average physician, medical ethics (of which there is no satisfactory exposition) means only medical etiquette and actually there is usually as great a penalty attached to a transgression of one as of the other. "Medical etiquette is concerned with the conduct of physicians toward each other, and embodies the tenets of professional courtesy. Medical ethics should be concerned with the ultimate consequences of the conduct of physicians towards their individual patients and towards society as a whole, and it should include a consideration of the will and motive behind this conduct."

Professional men have more specific moral issues confronting them than the mass experience of humanity has evolved rules to cover. This is especially true for the physician whose daily professional activities may often involve a choice between acting in the ideal interests of humanity or in the practical interests of self. Physicians have extremely vital relations with their patients, quite delicate relations with each other, and generally authoritative relations with the public. This complexity of responsibility, honor and prestige, tending as it does to exaggerate both the idealism and the egotism of the practitioner, makes it difficult for him clearly to see one path of righteousness, and to follow it alone. As a result, it is convenient for the doctor to find refuge in utilitarianism, the greatest good for the greatest number. This is an ineffectual compromise, for, as Fite has shown, it is really a calculated hedonism. Nevertheless, when one reviews the principle underlying the moral responsibility of medical men, as enunciated by physicians themselves, one feels that medical ethics is not based upon utilitarianism, but on idealism, primarily. From the Hippocratic oath to the latest revision of the Principles of Ethics of the American Medical Association, it is implied by all the medical ethical writers that the ideal ends in the interests of humanity, as the real basis for their remarks, and that these must be compromised only as little as possible in the interests of self.

New conditions are now arising in medical practice. No matter how greatly we may lament the fact, the old family doc-

tor, beloved of us all, is rapidly passing away in the growth of specialization, group-clinics, and general hospitalization of the sick. The close personal relations, between the physician and individual patient, are disappearing in the routine of technical diagnosis, mass treatment, and nationwide prophylaxis. Medicine is being recognized, more and more, as a matter for national and international public health effort, rather than as a profession catering to individual ambition. Its aim is its own end — the prevention of disease.

"Physicians and surgeons are occasionally requested to furnish certificates justifying the absence of persons who hold situations of honor and trust in the army, the navy, or the civil departments of government. These testimonials, unless under particular circumstances, should be considered as acts due to the public, and therefore not to be compensated by any gratuity. But they should never be given without an accurate and faithful scrutiny of the case; that truth and probity may not be violated, nor the good of the community injured by the unjust pretensions of its servants. The same conduct is to be observed by a medical practitioner, when they are solicited to furnish apologies for non-attendance on juries; or to state the valetudinary incapacity of persons appointed to execute the business of constables, church-wardens, or overseers of the poor. No fear of giving umbrage, no view to present or future emolument, nor any motives of friendship, should incite to a false, or even dubious declaration. For the general weal requires that every individual, who is properly qualified, should deem himself obligated to execute, when legally called upon, the judicial and municipal employments of the body politic. And to be accessory, by untruth or prevarication, to the evasion of this duty, is at once a high misdemeanor against social order, and a breach of moral and professional honor."

And so I leave with you these thoughts culled from Leake, Percival, Barker and others in the hope that this Academy, beloved of us all, will continue to go forward, and that during its progress, in spite of the changes in medical practice that will certainly present themselves as times change, we shall ever keep our ethical banner high, that we shall see to it that the superstructure of material medicine does not become too heavy for our ethical foundation, and so destroy it all, that we shall guard

well the grand tradition which is the heritage of this Society, and that each member of this Academy will ever continue to understand and to proclaim — "the duties of a physician."

> "Nay, come up hither. From this wave-washed mound
> Unto the furthest flood-brim look with me;
> Then reach on with thy thought till it be drowned,
> Miles and miles distant though the last line be —
> And though thy soul sail leagues and leagues beyond,
> Yet leagues beyond those leagues there is more sea."

Note — The material above dealing with Ethics was taken from "Percival's Medical Ethics," and "Introductory Essays" to "Percival's Medical Ethics" by Chauncy D. Leake, and "Manners and Morals in Medicine" by Lewellys F. Barker, Inter. Clinics, Vol. 11, Series 39, 1929.

SYMPOSIUM ON THE PROGRESS OF GENERAL MEDICINE IN RELATION TO EYE, EAR, NOSE, AND THROAT

I. HEMATOLOGY AND THE BLOOD DYSCRASIAS IN RELATION TO DISEASES OF THE EYE, EAR, NOSE, AND THROAT

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Hematology and the blood dyscrasias fully deserve a place, perhaps even the first place, in this symposium for progress in this field has been noteworthy and hematology has become a feature of the proper study of each and every patient. Unfortunately in the allotted time it is impossible to do justice to the subject — to describe its recent progress, to convey its current views and to emphasize the many points of practical usefulness. In this difficulty, I have compounded the following prescription for you:

℞

The normal and the abnormal blood picture . .	3 minutes
The origin of the symptoms of the blood dyscrasias	3 minutes
The individual dyscrasias	10 minutes
The hematology of infection	4 minutes

Sig.—To be well mixed, flavored with Tincture Otolaryngo-ophthalmologica, and swallowed rapidly at one dose.

1. *The normal blood picture* is maintained by a perfect balance between production and consumption of each cell type. Production proceeds under the control of stimuli about which we know little, and the various blood cells grow to an appropriate age in the shelter of their respective hemopoietic homes before entering the busy world of circulating blood. For this matura-

tion a whole series of stimuli, some hormonal, perhaps, seem to be necessary.

Proper maturation permits only sufficiently adult cells to appear in the blood and, if production and consumption are balanced there results what we accept as the normal bloodpicture. We have only recently, however, recognized that basal conditions are just as necessary for a constant blood count as for a metabolism determination. Wide variations in the white and red cell counts occur under the activities of daily life; the basal white count for example may double from muscular activity. This must be kept in mind in practice.

The other factor, destruction or consumption of the various cell types proceeds in different ways: erythrocytes are phagocytized by the reticulo-endothelium not only in the liver and spleen, but wherever this widely distributed tissue occurs; while the senile white cell is now believed to pass into the digestive tract chiefly through the mucous membranes of the mouth. This perhaps suggests that the constant involvement of the gums, mouth and pharynx in leucemia is analogous to the splenic enlargement of the anemias with increased blood destruction.

Little is known of the fate of the platelets; they probably are dissolved in the periphery of the body where their potent juices remain, to supplement the circulating platelets in time of need.

The abnormal blood picture results from any disturbance of the balance — the so-called hemolytopoietic balance — between production and consumption, or from an added element of abnormality — often termed "degeneration."

Production may be decreased or speeded up, and maturation may be interrupted. Such an arrest of maturation best explains the pathology and blood picture of Addisonian anemia and an analogous arrest may be found to explain other important disorders of the blood; for example, the leucopenia which may lead to agranulocytic angina. Progress will come as we learn the successive stimuli required for normal maturation of the erythrocyte, the platelet and the various leucocytes.

2. The second ingredient in our prescription concerns the common origin of many of the symptoms seen in the group of blood dyscrasias.

No doubt each of the blood dyscrasias is an entity, but certain basic changes in the blood recur frequently in their various blood pictures. Anemia, leucopenia and thrombocytopenia, for example, are present as primary and secondary changes in many of the dyscrasias, and whenever present are likely to lead to the same symptoms. For example, a low platelet count explains the retinal hemorrhages of such widely different dyscrasias as acute leucemia, aplastic anemia and purpura hemorrhagica. Also tinnitus and pallor may occur in any dyscrasia in which anemia is part of the blood picture.

It is most important for you to realize the disease non-specificity of such symptoms and to relate them to the basic change in the blood picture rather than to any one clinical syndrome.

Also certain tissue changes may occur in more than one dyscrasia and may cause identical results. For example, tissue infiltration with abnormal white cells is common to the various leucemias and may by pressure, cause deafness or other local symptoms. Any leucopenia opens the way to infection.

Of course, local differences in anatomy and physiology influence very much the distribution of such manifestations. Such local determinants are not the same in the ear as in the mouth, nor the same in the eye as in the tonsil. The mouth and the colon as frontiers constantly exposed to bacterial invasions, have more in common than the mouth has with its neighbors, the eye and the ear. One is, therefore, not at a loss to understand why the same necrotic lesions often occur in mouth and colon in agranulocytic leucopenia and seldom, if ever, in ear or eye.

Furthermore, many of the manifestations of the blood dyscrasias are determined by local circulatory conditions. For example, the peculiar vascularity and visibility of the retina permit the recognition of capillary hemorrhage and of the pathognomonic pictures of polycythemia, intense anemia and leucemia. In contrast, the structure of the ear renders its vessels and nerves very liable to pressure from hemorrhage or infiltration both of which occur in Addisonian anemia, purpura hemorrhagica and, in leucemia, especially into the labyrinth.

3. Let us next comment on certain of the dyscrasias — first the anemias. Only a few years ago an authoritative text on medical ophthalmology discussed at great length the eye symp-

toms of chlorosis, but gave little or no attention to other forms of anemia. Today we seldom see chlorosis and we realize that the ocular manifestations then described for chlorosis are those of any anemia.

Chlorosis was probably due to a faulty dietary and more and more we appreciate the part played by disturbance of the digestive tract in the production of the anemias. In the Addison form the lack of a specific intrinsic factor in the gastric juice independent of hydrochloric acid or pepsin, leads to a deficiency in a substance necessary for the normal maturation of erythrocytes. Similarly, most if not all of the anemias resembling the Addisonian type, have a distinct relation to the digestive system, for example, the anemias of sprue, fish tapeworm infestation, cancer of the ascending colon and certain intestinal strictures.

The interest which formerly centered on the spleen as the mysterious cause of anemia has been transferred to the gastrointestinal tract.

Of course, in any discussion of anemia, it is the treatment of Addisonian anemia which overshadows all other progress; no longer can this disease be called either primary or pernicious. There is no more fascinating story of progress than that of the steady march forward from the use of raw liver to today's highly potent extract for parenteral injection.

Early diagnosis and proper treatment will prevent in the future the fully-developed picture of Addisonian anemia. No longer will we see the characteristic atrophy of the tongue nor the horrible tragedy of posterolateral sclerosis which even liver will not cure. If this complication ceases to occur, I am afraid that most clinicians who still keep tuning forks only to test for the characteristic loss of vibratory sense will put them away in the cabinet with the discarded monaural stethoscope and the cups.

Concerning other anemias little need be said. Sickling of the red cells, that peculiar change of shape which occurs in wet preparations, has been found to be more common than was at first suspected and often to occur without anemia. It is clearly hereditary and almost limited to the Negro race. It is important for this reason to distinguish another familiar symdrome with oval erythrocytes, which occurs in others than Negroes and without anemia.

Perhaps you have encountered the latest fashion in classification of anemia; that based on the size of the red cell and its hemoglobin content. Anemias are normo, macro, or microcytic and if the hemoglobin saturation is low, hypochromic; indices based on cell volume are to replace our old friend the color index. This classification leads to diagnosis such as "chronic hypochromic anemia with achlorhydria and microcytosis." This syndrome resembles chlorosis, but occurs in middle aged women and may be analogous to the leucopenia of agranulocytic angina which is most common in women of that age, and to a form of thrombocytopenia described by Nagy in women with ovarian dysfunction.

There is nothing to recount of the polycythemias; the frequency of visual symptoms and the characteristic retinal picture have been brought home to us by emphasis in the literature.

The leucemias have been the subject of little advance in knowledge. We are still totally ignorant of the cause of any of the various types, but we do perhaps realize a little better that they are very different diseases with nothing in common other than the white cell increase. The renewal of therapy with arsenic has not led to any better results than were obtained many years ago.

There has been much recent interest in "leucemoid" blood pictures, such as occur in infectious mononucleosis, in the pseudoleucemic anemia of infants, and in certain unusual reactions to infection best seen perhaps in whooping cough.

The similarity to leucemia rests either on a marked increase in the total white count, on the presence of many immature cells, or on some other change from the normal picture. In contrast to true leucemia the leucemoid blood picture can usually be related to a definite cause and is only temporary. The term "leucemoid" is also employed of another group of conditions which find their resemblance in tissue pathology without change in the blood picture. Chloroma, familiar to the ophthalmologist from the intraorbital tumor deposits, is an example.

Leucopenia is probably the disorder of the blood which at this time is foremost in the minds of many of you, because of its relation to agranulocytic angina.

It has long been known that leucopenia from a reduced number of neutrophils occurs in some infections, including over-

whelming infection; in aplastic anemia, in benzol poisoning, sometimes after arsphenamin, and during anaphylactic shock. Hematologists were slow, however, in recognizing a primary apparently causeless, form of leucopenia analogous to primary anemia and to primary purpura hemorrhagica. It is now realized, however, that in mild degree this is fairly common and may be associated with no symptoms beyond perhaps a tendency to fatigue. In some individuals the leucopenia is chronic, in others it comes and goes for years, sometimes in quite regular cycles. Only when the leucopenia is severe do the amazing non-reactive necrotic lesions appear in the mouth, pharynx, bowel, vagina or skin. At first the cart was placed before the horse, but we now know that the leucopenia is primary and the infection secondary. We do not know, however, the cause of the leucopenia; there is no evidence to relate it to inhalation of exhaust fumes, to chronic poisoning from cans or other food containers, to endocrine dysfunction, all of which have been suggested. Several of my patients with agranulocytic angina were allergic and this may play a part in bringing about the leucopenia just as allergy probably explains the relative lymphocytosis occurring from neutrophil decrease in sympathetic ophthalmia.

Whatever the cause, we have not yet at our disposal any satisfactory treatment of primary leucopenia. There is some evidence that an arrest of maturation is the pathogenesis but we know neither the lacking stimulus nor how to supply it. Transfusion has been the therapy of choice, but today nucleotide injections are being widely employed apparently with some success. Unfortunately the response is not uniform nor prompt enough to save the fulminant type of case which is that giving the greatest mortality.

Next a word about the purpuras — not to describe progress — but to admit ignorance. Purpura appears in so many diseases that the phenomenon is no more specific than is anemia. Again we find primary and secondary forms. Rheumatic purpura is merely a symptom of the infection and a coincident iritis is not to be blamed on the purpura but on a common background as was emphasized by Benedict[1] in 1930. Any focus of infection may cause both purpura and arthritis as in the patient reported by Borries[2] with antral empyema.

Even when we take into account quantitative and qualitative disturbances of platelets, and deficiencies in calcium and fibrinogen, we are unable to offer a satisfactory classification of the hemorrhagic disorders. That the platelets do not explain the whole matter has recently been strongly stressed by Morawitz.[3]

Normally there are sufficient platelets to plug any tears in the vascular endothelium and to supply the material essential for coagulation. When thrombocytopenia is present a purpura is readily attributed to the lack of platelets, but so often purpura occurs in the presence of a normal platelet count, as to leave the whole subject in confusion. There is great need of further progress in this field.

4. *The Hematology of Infection* — Hematology does not find its field of greatest practical value in the identification of rare blood dyscrasias, but rather in the study of disorders encountered daily by the physician, the surgeon and the specialist. Sometimes the change in the blood picture is spectacular as for example the stippling of erythrocytes in lead poisoning, the lymphocytosis of whooping cough, the eosinophilia of trichiniasis and the monocyte increase of subacute bacterial endocarditis.

Far less spectacular, but of much greater importance for all of us, are the changes which accompany infection. Interest has progressed from the increase in the total white count and the neutrophil percentage to the appearance of immature cells. This latter sometimes gives the so-called leucemoid blood picture but more significantly it gives an opportunity to gain insight into the presence, progress and prognosis of an infection from the relative ages of the neutrophils present.

Arneth in 1904, using the nuclear configuration as a basis, tabulated the neutrophils in columns according to age. His method as well as Schilling's even more complex "hemogram," have been almost abandoned for routine use — but we still speak of a "shift to the left," indicative of an increase in young forms, and a "shift to the right" of old.

Simpler and more practical methods merely divide the neutrophils into two age groups according to whether the nucleus has or has not reached the stage when the chromatin segments are connected by mere filaments. Normally the cells with non-filament nuclei average only eight per cent of the neutrophils

and the upper limit in normal blood is sixteen per cent. An increase above these figures is a "shift to the left" and indicates an increase in immature forms. This may occur in the absence of any increase in total count and even without any distinct increase in the neutrophil percentage. Under such conditions the method may give very useful information as to the presence of infection, but only repeated observations permit one to draw many conclusions.

A word of warning is necessary; even the simplest of these methods requires care and skill. We must be on our guard not to allow dogmatic reports from the laboratory to lead us to neglect other clinical evidence and our own practical judgment.

Finally we must mention the usefulness in infection of the determination of the sedimentation rate of the erythrocyte.

Many years ago during the period when phlebotomy was so copiously performed, one of the main indications for bleeding was an increase in the buffy coat, or as it was called, the "size" of the blood, which depends in a large measure on the rapidity with which the erythrocytes settle before coagulation occurs.

After bleeding became unfashionable, there was no further interest in this until recently an increased rapidity of sedimentation has been found to have a peculiar value under certain conditions of infection. The test is in no way specific; an increased rapidity is present whenever there is tissue destruction as in all infections, in fever, with tumor cachexia and in pregnancy.

In spite of this the test has proved of great value in determining the continuance of infectious activity, especially after fever, leucocytosis and local signs subside, as, for example, in pelvic inflammatory disease, pulmonary tuberculosis and rheumatic infection. It seems likely that there are similar uses for this method in your specialties.

Conclusion — There is much more that should be said about hematology, but time allows only a closing paragraph.

Hematology owes its progress to research and to elaborate methods but in practice the methods of hematology must be such as we can perform, and the information derived must be such as we can understand and apply. You and I, in our daily work,

must know the tools we use and what we can count upon them to do for us. We must not be easily led to abandon the tried and true for something merely new, but we must be ready to hear of the new and adopt it if its value is proved. That the members of your Academy are thus open-minded and progressive is evidenced by this symposium on your program in which it has been my pleasure and honor to take part.

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II. CARDIOVASCULAR DISEASES IN RELATION TO THE RETINA

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LIEBREICH, in 1859, gave the first accurate description and graphic illustration of "albuminuric retinitis." Twelve years later, or sixty years ago, Allbutt, who was primarily an internist, published a book on the use of the ophthalmoscope in certain diseases including those of the kidney. Allbutt discussed the possible relationship of the retinal changes to renal pathologic processes. He admitted the difficulties in explaining such a relationship, but definitely stated that in his opinion the retinitis occurred late in the course of the renal disease and was of serious prognostic import. Michel, in 1884, was the first authority to state how long a patient might be expected to live after the development of albuminuric retinitis. His own statement was: "In regard to the maintenance of life, the retinitis is of very serious significance; in the majority of cases in which simultaneously retinitis appears and the diagnosis of granular kidney is made, after a course of one-half to one and one-half years, death can be expected."

Physicians are indebted to Marcus Gunn, in 1898, and to Foster Moore, in 1916, for distinguishing the arteriosclerotic changes in the retina from those associated with primary renal disease. They were both of the opinion that certain retinal arterial changes were localized lesions of a general arteriosclerotic process occurring throughout the body. Moore showed that there was a relationship between the increase in systolic blood pressure, and the extent of the retinal arteriosclerotic changes. O'Hare and Walker, in 1924, pointed out clearly that hypertension exists, or has existed, in any case in which there are sclerotic changes in the retinal arterioles. From this they assumed

that the condition of the retinal arterioles is an index of the state of the arterioles of the tissues generally.

In October, 1920, I became very much interested in the subject of albuminuric retinitis. A young man was admitted to the hospital with a diagnosis of chronic glomerulonephritis. Both Dr. Wagener and I were convinced that the patient had typical albuminuric retinitis, but much to our surprise his renal function was extremely good, much too good for that seen in cases of terminal chronic diffuse nephritis. This patient was our first recognized case of so-called malignant hypertension. By further clinical, ophthalmologic and pathologic studies, we were able to identify a syndrome, which was characterized by hypertension and diffuse arteriolar changes throughout the body. The prognosis was serious, and depended in large part, on the type and extent of the retinal changes. By this work we were led to distinguish, from general and retinal arteriosclerosis with essential hypertension, a special group of cases of late and rapidly progressive diffuse arteriolar disease.

The pathologic changes in the arterioles that have been mentioned usually have been considered organic in nature. The significance of vasospasm without preceding organic changes has recently been shown by Haselhorst and Mylius in eclampsia. They actually demonstrated in a case of eclampsia, by retinal photographs, the presence of arteriolar spasm, and the rapid disappearance of the spasm following delivery. In the present year, Koenigsberger and Bannick reported a case, in a male subject, of acute vasospastic hypertensive disease, in which later, so-called malignant hypertension developed. In the early stages in this case, accurate retinal studies were not made. Subsequently, Wagener, Burke and Barker described a small series of similar cases in which vasospasm, including that of the retinal arterioles, was a prominent feature. The arterioles of the skeletal muscles in these cases were shown to have intimal proliferation. Therefore, these authors expressed the belief that both vasospasm and actual pathologic changes involved the arterioles throughout the body.

For the understanding of the relationship of changes in the retina to cardiovascular disease it is necessary to consider the association of hypertension with arteriolar spasm, glomerulonephritis and diffuse arterial disease.

Essential Hypertension Associated With Arteriolar Spasm

Several observers, including Elschnig and Wagenmann, have recorded transient blindness associated with occlusive spastic constriction of the retinal arterioles. With the quick return of vision the spasticity of the retinal arterioles disappeared. A similar case has been followed by Dr. Wagener. Numerical data concerning this and other cases referred to in the paper will appear, for the most part, in the table. The history and general data will be given in the text.

Case 1—A physician's wife, aged forty-three years, gave the history that several years before she had had two miscarriages, with which there had been associated albuminuria. Also for four or five years she had had attacks of petit mal, for two years, white or dead fingers, and for ten years, short periods of partial or complete blindness. At our initial examination, we thought the visual disturbance was probably secondary to general spasm of the cerebral vessels, rather than of the retinal vessels, for moderate essential hypertension was present, accompanied by slight sclerosis, but by no visible spasm of the retinal arterioles. The attacks of petit mal and white fingers were also considered to be due to arterial spasm. While the woman was being examined again, two years later, Dr. Wagener observed with the ophthalmoscope, actual occlusive spasm of the left inferior temporal artery as an attack of blindness occurred. The spasm and blindness persisted for one or two minutes and then the previous conditions were restored. This observation indicated that the temporary blindness was due to retinal vascular spasm and not to cerebral spasm, as we had previously incorrectly thought. Four years later, thrombosis of a retinal vein (superior temporal) developed.

The probable sequence of events in this case were the development of eclampsia with a residual tendency to hypertension and recurrent arterial or arteriolar spasm. The significant fact which should be stressed is that arterial spasm can occur in the retina and tissues generally for a short period and leave no demonstrable injury of tissue. If, however, the spasm persists, an anemic infarct results with permanent loss of vision.

In eclampsia, vasospasm, although not completely occlusive, is an important factor. If it persists long enough, ischemia or venous stasis with secondary organic lesions of the retina develop. Many authorities presume that vasospasm is the initial basis for the renal pathologic changes in this condition. Yet, if the eclamptic condition is relieved by delivery, the vasospasm may cease and complete restitution of the retina and kidneys may take place; on the other hand, even after delivery, vasoconstriction may persist and the retinal and renal lesions progress. In the case of the physician's wife just reported, presumably there was a residual tendency to periodic vasospasm. In those cases in which vasospasm persists the primary abnormal physiologic process probably gradually gives rise to pathologic changes in the arterioles. That such a sequence of events may occur during, and subsequent to, eclampsia seems logical judging from facts known at present. A similar sequence of vasospasm followed by pathologic changes would at least offer a good explanation for the facts observed in the series of cases reported by Koenigsberger and Bannick, and Wagener, Burke and Barker, in which the patients were males and nonpregnant females. Next is the example of such a case.

Case 2—A man, aged thirty-five years, gave much direct and indirect evidence of diffuse hypertensive arterial disease. He had had hematuria for two and a half years, hypertension for one year, and headaches and visual disturbances for one year. The retinal arterioles were small and evidently constricted. Arteriolar thrombosis was demonstrable in the retina. On histologic examination of the arteries and arterioles of muscle tissue obtained for biopsy, there was definite narrowing of the lumen, pathologic changes in the wall, and, in addition, actual arteriolar thrombosis. Thus, it was possible to demonstrate by the ophthalmoscope and by histologic technic that arteriolar spasm coexisted with actual anatomic changes, and that arteriolar thrombosis had occurred. The right renal pelvis was found to be dilated and moderate secondary anemia was present. The patient died within a few months.

This case demonstrates how serious the prognosis may be. On the other hand, possible relaxation of the arteriolar spasm may explain the fact that in certain cases of well-developed vascular retinitis remissions do occur.

Glomerulonephritis

It is now generally recognized that retinal changes occur only in a small percentage of cases of definite diffuse glomerulonephritis. This fact has become more apparent in the last few years, with the recognition that diffuse vascular disease rather than renal disease is the cause of many cases of so-called albuminuric retinitis. Retinitis is extremely rare in acute glomerulonephritis, and the few instances studied indicate that the findings are similar to those present in eclampsia. In subacute glomerulonephritis, general edema is often a troublesome symptom. It is in such cases that retinitis characterized by edema of the disk, large "snow-bank" exudates and even detachment of the retina occur. Also, this type of retinitis is seen during an acute exacerbation in the course of chronic glomerulonephritis. In this type of retinitis, edema is a prominent feature and apparently is a local manifestation of the general disturbance in metabolism of water and salt.

Case 3—A woman, aged twenty-seven years, gave the history that when she was twenty-three years of age she had albuminuria and edema, and infected tonsils were removed. Three weeks before I saw her she had a respiratory infection followed by edema. Two days after her admission, she had acute pericarditis.

Two retinal lesions occur late in the course of chronic glomerulonephritis: angiospastic (albuminuric) retinitis and that associated with chronic anemia. The former is very similar to the retinitis of diffuse arterial disease, but changes in the vessels are less marked.

Case 4—A man, aged nineteen years, had had edema for two months before I saw him, and at that time mild secondary anemia was present. About a month after I examined him the edema had disappeared, and tonsillectomy was performed. But the edema returned and his condition became worse. On the last two days of his life, approximately eleven months after I first saw him, and thirteen months after the beginning of his illness, he was markedly edematous and anemic, and convulsions occurred. Angiospastic retinitis was present in the last six weeks. Necropsy revealed chronic glomerulonephritis.

The present conception as to the cause of "albuminuric retinitis" is that it is secondary to arteriolar changes, but that these are less marked than in chronic diffuse arterial disease. They are probably primarily angiospastic rather than angiosclerotic. The presence of this type of retinitis is of very serious prognostic significance.

In cases of chronic anemia due to different etiologic factors, characteristic retinitis can occur. Such retinitis may be present in the terminal phase of chronic glomerulonephritis. If, in addition to this retinitis, there are changes in the arterioles, renal or diffuse vascular disease should be suspected, and, if possible, excluded. In case five there was such a type of retinitis.

Case 5—A woman, aged fifty-nine years, was first seen in 1922. In 1929 she had an adenoma of the thyroid gland, with basal metabolic rate of —6, pains in the lower part of the legs, and dyspnea on exertion. In 1932, secondary anemia was present, and she had had attacks of loss of consciousness for one year. There was 7.4 gm. of hemoglobin in each 100 cc. of blood and erythrocytes numbered 3,480,000 in each cubic millimeter of blood. She died later in that year. Necropsy revealed hypertrophy of the heart. The kidneys, of which there was arteriosclerotic atrophy, had a combined weight of 75 gm. Acute pericarditis, also, was present.

Essential Hypertension Associated With Diffuse Arterial Disease

Allbutt, in 1896, clearly described the clinical syndrome of essential hypertension and termed it "hyperpiesia." He recognized that such a condition is compatible with continued good health; indeed, he had observed one of his patients for twenty years. Luckily, at the present time this group of cases constitutes the great majority of those of hypertension and I have termed it "diffuse arterial disease with hypertension, group one."* A study of blood pressure for twenty-four hours in one of the cases seen at the clinic (case six) demonstrated the decided effect of rest on the hypertension. The retinal changes in this condi-

* The grouping of these cases into four groups has been an aid in estimating prognosis. Each group has distinct features which may or may not prove to belong to a single disease process. Further study, I hope, will confirm the usefulness of this grouping.

tion are minimal and consist of slight but distinct organic narrowing of the arterioles.

Case 6—The patient was a man, aged thirty-eight years when first seen in 1918. At that time he complained of having had lumbago for five weeks; he also had cardiac arrhythmia. In 1924 the cardiac arrhythmia was still present. He did not have hypertension either in 1918 or in 1924. In 1930 the man was fifty years of age, at which time he had hypertension and headaches, and had noticed increased irritability for two years. He had had an attack of vertigo one month before his return for study. One year, and also two years later, he felt well.

Patients who have a continuously higher blood pressure and more distinct anatomic changes in the arterioles than those of the previous group, but who still are in good general health, have been placed in a second group, called "diffuse arterial disease, group two."

Case 7—In 1928, a woman, aged twenty-three years, was known to have had hypertension and headaches for six months. Arterioles of the muscles were found on histologic examination, to be definitely narrowed. Although she has diffuse arterial disease, her health has remained good for four years.

One's impression of these patients of group two is that the disease is of a more serious nature, either due to a more rapid course since the onset, or because they have a later stage of the condition described under group one. Follow-up studies in this entire group show that the prognosis is more serious than in group one.

Group three includes cases in which there is hypertension and readily demonstrable diffuse arterial changes, but in which, in addition, there is mild retinitis. Case eight is a good example of the progressive type of diffuse arterial disease. As a group the prognosis in these cases is serious.

Case 8—A woman, in 1929 was aged thirty-five years, and at that time was known to have had hypertension for six months. Three years later, headache and pain in the legs had developed. Within these three years, the condition had changed from that of group two to that of group three. Studies of blood pressure, as well as retinal changes, indicated that the course of the disease was rapid and progressive.

It is of great interest that even in cases of group three there may be a remission in the activity of the pathologic process, as in case nine.

Case 9—A woman, aged thirty-one years, was seen by me for the first time December 12, 1931. She had edema of the eyelids, and knew that she had had hypertension nine years before. Three years before I saw her she had a nasal hemorrhage, and one year previous to this, right hemiplegia appeared. On ophthalmoscopic examination there was evidence of a previously active process and many small arterioles were seen to have become obliterated. Some arterioles of the retina had undergone thrombosis, and on biopsy the same process was seen to have taken place in some arterioles of a skeletal muscle.

Could cessation of arteriolar spasm be responsible for the remission in this case nine?

The use of the term "malignant hypertension" has been adversely criticized by many physicians. To avoid any controversy over terminology, I have grouped such cases under the title of "diffuse arterial disease with hypertension, group four." These undoubtedly are the most serious examples of this disease. In the first description of these cases, the prognosis was said to be very serious and the duration of life to be only a matter of months or a year or two. Subsequent studies of a much larger series of cases has confirmed this impression, and led me to the conclusion that the similarity of Michel's statistics in 1884 to mine indicates that the majority of his cases of "albuminuric retinitis" belonged to this group four. The characteristic findings are diffuseness of arterial and arteriolar thickening throughout the body, and the retinal changes. Wagener has emphasized the marked spastic and organic narrowing of the arterioles and the edema of the disks. The presence of the latter he considers the feature that distinguishes this type of retinitis from the retinitis seen in cases of group three. Case ten is an example of a rapidly progressive condition.

Case 10—A man, aged thirty-seven years, when first examined in March, 1932, was up and about. He had had hypertension for two years and headache for eighteen months. Gross hematuria, which had appeared ten days before, caused him to come to the clinic. Slight failure in vision had been noticeable for one year. The appearance of the ocular fundus was typical

of cases of group four and diffuse arterial disease was evident. One month later, diffuse pain in the muscles appeared, and then came blurring of vision of the left eye. About two months after I first saw the patient he died from cerebral, cardiac and renal insufficiency. Necropsy revealed hypertrophy of the heart, ischemic nephritis and acute pericarditis.

Not all cases of group four are of such rapid course. Some patients even have periods suggesting remission, such a case, belonging to group three, was considered previously. Here again spasm of the arterioles may determine whether the condition is progressive. I have observed a few cases of group four, without histologic changes in the arterioles of the skeletal muscles. Evidence is also accumulating that even when there is marked pathologic change in the arterioles, compensatory mechanisms may still permit periods of improved tissue metabolism.

Comment

Since Helmholtz devised the ophthalmoscope eighty years ago, knowledge regarding vascular changes in the retina has steadily increased. Studies of the ocular fundus have played a significant part in the gradual definition of diffuse arterial disease and in its separation from primary diffuse glomerulonephritis. The actual observation of the normal and abnormal retinal arterioles during life, and the knowledge that arterioles in the tissues everywhere may be reacting similarly, have given new facts and stimulated new ideas as to the physiologic function of, and pathologic changes in arterioles. Abnormal vasospasm can be observed in the retinal arterioles; when of very short duration no gross changes occur in the retina; if prolonged, secondary retinal lesions develop, and there is evidence that prolonged vasospasm may lead to histologic changes in arterioles. It has been possible to show that vasospasm and definite histologic changes can coexist. In such a case, as one might expect, there is a tendency for thrombosis to occur in the arterioles. Relaxation or intensification of the arteriolar spasm offers a possible explanation for the remissions and exacerbations that occur in the course of diffuse arterial disease.

The type of retinal vascular changes, and the presence or absence of retinitis, are important aids in arriving at prognosis. At present the conception of the end stages of vascular retinitis

is much clearer than formerly. Nevertheless, when serious lesions of this type are observed in the retina, Liebreich's experience in 1859 should be recalled. He observed complete subsidence of a marked "albuminuric retinitis."

There are still many factors in the course of diffuse arterial disease that are difficult to explain. The etiology is unknown. Cases in which the course is rapid and progressive are in distinct contrast to those in which health is good for many years. There is no satisfactory explanation of why a few patients belonging to group four have competent peripheral arterioles in certain tissues, whereas those in the retina are thickened. Therefore, much investigative work has still to be carried out to unravel these problems. The combined efforts of ophthalmologists, internists and pathologists have already made notable advances in the knowledge of these obscure diseases and the results so far attained have been a great stimulus for these workers in different branches of medicine to continue their joint efforts.

Summary

Studies of the ocular fundi have played a significant part in distinguishing primary, diffuse arterial disease from diffuse glomerulonephritis. Observations of the normal and abnormal retinal arterioles during life have added to the knowledge of the physiology and pathology of arterioles throughout the body. Spasm of the arterioles may or may not lead to retinitis. When spasm coexists with demonstrable histologic arteriolar lesions, retinitis and thrombosis result. Relaxation or intensification of arteriolar spasm may explain the remissions and exacerbations that occur during the course of diffuse arterial disease. Such studies suggest the possibility that there are peripheral compensatory mechanisms when even serious arteriolar lesions exist. Retinal vascular changes are of practical significance in prognosis.

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TABLE I — *Summary of Cases**

Case	Date	Blood pressure	Pulse rate	HEART			RETINA			URINE				RENAL FUNCTION		COMMENT
				Size, grade	Electrocardiogram	Peripheral sclerosis, grade	Sclerosis, grade	Retinitis, grade	Vasospasm, grade	Specific gravity	Albumin, grade	Casts, grade	Erythrocytes, grade	Blood urea mg. for each 100cc.	Phenolsulphonphthalein, per cent	
1	12- 5-23	160/115 140/ 85	74	1		1	1	0	1	1.018	1	1	0	16	45	
	9-29-24	170/120 160/115		1			1	0	1	1.017	0	0	0	17		
	4-13-26	160/100 155/100		1		2	1	0	3	1.015	2	0	0		45	Spasm of artery observed
	8- 8-30	150/110 155/110					2	1								Thrombosis of retinal vein
2	10-27-31	220/110	75	1–2	**	3	See Text			1.008 1.017	2–4	1	4	28	30	
	10-31-31	200/140	66													
	2- 1-32															Died
3	3-23-32	150/105 120/ 90	110	1	0	2	0	3	1	1.009	4	0	2	222		
	3-25-32													222		Acute pericarditis
4	12-28-25	180/100 130/ 80	100	1	0		0	0	0	1.009 1.020	3–4	1–3	1–2	50	50	
	1-22-26									1.018	3–4	0–2	2	78	45	Tonsillectomy
	6- 2-26	160/100 135/ 80	80	1		1	0	0	?	1.012 1.025	1–2	1	1	56	50	Moderate edema
	6-19-26													74		
	10-13-26	180/110 160–100	80	1		1	0	1	?	1.007 1.025	1–3	0–1	0–1	59	50	Slight edema
	11-27-26	180/120	115	1		1	0	2						177		
	11-29-26															Died

5	9-28-22	145/ 90								1.015	1	0	0			
	7-18-29	165/ 90	96		0		1	?	0	1.008	2	0	0			
	6-20-32	220/110 150/ 90	90	1	0	3	2	1	0	1.011	2	0	1	126		
	6-27-32	160/ 90	90								2	0	0	118	6	Blood creatinine 7.2 mg. in each 100 c. c.
	7- 3-32															Died
6	7-16-18	120/ 80	72				0	0	0	1.029	0	0	0			
	7- 9-24	130/ 80	72	0	0					1.024	0	0	0			
	11-17-30	210/130 140/ 90	75	1	0	2	1	0	1	1.003 1.027	0–2	0	0	32		
	7-21-31	170/100 160/ 90	68	1		2	1	0	0	1.015	0	0	0	16		
	7-25-32	200/130 170/120	70	1		2	1	0	0	1.027	0	0	0	30		Well
7	5-19-28	205/160 140/115	85	0	0	0	1	0	1	1.001 1.028	0	0	0	15	75	
	4-23-31															Condition good
	8- 6-32															Good health
8	5- 6-29	230/145 150/ 90	80– 100	1	0	1	2	0	1	1.001 1.031	0–1	0		19	50	
	6- 6-32	260/160 195/125	80– 110	1	***	2	2	2	1	1.020	3	1		32		Condition worse
9	12-21-31	260/175 175/125	95	2		2	2	?	0	1.003 1.016	0–2	1	0	18	35	
	12-30-31									1.016	2	0	0		45	Condition improved
10	3-18-32	220/160 170/110	85	1	***	2	3	2		1.005 1.035	3–4	1	1–4	38		
	5- 4-32	190/130 140/ 90	90 120	1	***	2–3	3	2		1.011 1.019	3–4	1–2	1–4	80		
	5-21-32													232		
	5-26-32															Died

* Wherever the symbol zero appears, it means that no abnormality was present although an endeavor was made to detect one; wherever a space is blank, it means that a test, procedure, or examination was not carried out.
** Inverted T wave in deviations 1, 2 and 3.
*** Electrocardiographic changes present.

III. OPERATIVE TREATMENT OF MIGRAINE AND OBSERVATIONS ON THE MECHANISM OF VASCULAR PAIN

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MY UNDERSTANDING of this symposium is that those of us who are wont to worship in other temples are permitted to bring votive offerings from afar to the shrine of the special senses, a shrine where the eye, the ear, the nose and the throat has each its own peculiar altar.

If this is true I may perhaps be allowed to begin my discussion of head-pain by a brief consideration of referred pain in general and of angina pectoris in particular.

When a pathological process takes place in the gall bladder impulses pass upward through the sympathetic nerves and the sufferer feels pain in the shoulder as though one of the cutaneous branches of a thoracic nerve had been stimulated. Another sufferer who has an abnormal condition in a coronary artery is tortured with pain in the left arm or in the skin of the precordium, not in the heart.

This has been named referred pain (James Ross) but there are sometimes obvious objections to the classical explanation given to this phenomenon by Head and MacKenzie. First of all no direct anatomical pathway has been found from the sympathetic system into posterior roots and the pain tracts of the spinal cord. Secondly, if novocaine be injected into the area of skin to which a sufferer refers his cardiac pain, "mirabile dictu," the pain disappears. Therefore, it would seem that through that area of skin runs the neurone chain which bears the agonizing impulse to the brain. The pathway would lead them in this case from the heart along the sympathetic fibers to the periphery; from there it would travel into the central nervous system along the ordinary cerebrospinal nerves and into the spinal pain tracts by a familiar route.

As the result of studying a series of operative cases of angina pectoris (Penfield, 1925), I made the suggestion that the pain in this condition was not produced by afferent conduction up sympathetic nerves to the spinal cord, but that the irritating impulse passed up a sympathetic axone to be discharged outward along collateral branches, producing a condition at the periphery (e.g., in the skin) which gave rise to painful impulses in the ordinary cerebrospinal nerves to that part. The afferent impulses up these nerves then gave rise to the referred pain. Such a diffusion of an impulse is called an axone reflex.

The same conclusion has been reached independently by others, but most convincing experimental proof has just recently been brought forward by Davis and Pollock (1931). After careful experimental section of various nerves, in turn, they conclude that the sympathetic nervous system carries efferent impulses which produce "a possible metabolite" which stimulates the general cutaneous sensory endings. The painful impulses which result are carried by the somatic afferent system through the posterior roots. After cutting all of the highest twenty-three posterior spinal roots in a cat they found that stimulation of the superior sympathetic ganglion still produced pain but that section of the posterior root of the fifth immediately abolished this pain. In other words, complete section of all adjacent cerebrospinal nerves blocked entrance of any pain impulse from sympathetic into spinal cord.

Time does not permit further elaboration of this theme. At least certain types of visceral or vascular pain depend for their existence upon an axone reflex (Langley and Anderson, 1894) that takes place within the sympathetic nervous system. Thus an impulse may pass antidromically up an efferent sympathetic fiber and be passed to many other ganglion cells by means of the numerous collaterals belonging to the originally stimulated axone, or doubtless a post-ganglionic collateral may be stimulated locally so as to cause the motor discharge into all of its numerous other collateral branches (Fig. 1).

Without discussing the nature of axone reflexes here the purpose of this presentation will be served if it is remembered that isolated sympathetic neurones *may spread the effect of a local abnormal process,* so that it is felt as pain through cerebrospinal nerves that innervate distant as well as adjacent parts. The following case illustrates such a spreading effect.

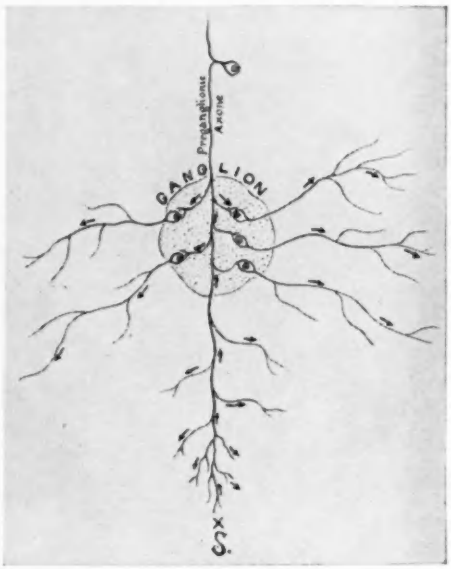

FIG. 1—Diagram to illustrate the probable pathway of impulses in an axone reflex. The stimulus arises at *S* and passes up the axone, sending an outward impulse along the collaterals to be distributed widely to the periphery. That such a process may take place in a post-ganglionic as well as a pre-ganglionic axone seems likely, but awaits proof.

The patient (F.H.) was a woman in her forties who had been suffering from angina pectoris for two years. She was under the care of Dr. A. Strong of New York, who found no objective evidence of heart disease either by electrocardiogram or by physical examination. She had been an unusually active woman until two years previously when, at the end of a thirty-mile walk in the country, she noted precordial pain. This increased until she was unable to climb stairs without the most severe feeling of compression in the precordium and left arm. Emotional disturbance produced even more severe seizures. Nitro-glycerine and complete quiet afforded the only relief.

Six months before the onset of the pain a swelling had appeared in the left side of the neck. We were struck by the possible relationship of the swelling to the anginoid pain, but I postponed exploration as long as possible, perhaps because the patient was a close personal friend of mine! Finally in February, 1927, I explored the neck. The mass proved to be made up of tuberculous lymph glands. Over the surface of the mass and

closely adherent to the general capsule was the cervical sympathetic chain. The gland was removed and the sympathetic chain at that point severed. No ganglia were disturbed.

From the time of operation there have been no more attacks of angina pectoris and I learned that the following summer she climbed a mountain against advice. She explained this failure to heed professional warnings by saying that it seemed to her to be only a small mountain!

It is now nearly five years since operation and she has continued in perfect health. The mechanism here was obviously irritation of the sympathetic chain by the enlarged gland resulting in antidromic impulses to the stellate ganglion. From these the impulses were reflected to the peripheral parts to which stellate fibers pass. At the periphery the impulse was transferred to cerebrospinal nerve endings.

Perhaps it may seem a far cry from angina pectoris to migraine and head pain, but I have made this devious approach in the belief that analogous mechanisms underlie angina and migraine, and that our failure to cure the latter condition may well result from failure to appreciate its nature.

FIG. 2—Nerve ending in adventitia of pial artery.

Intelligent application of any hypothesis of the mechanism of head pain demands preliminary inquiry into vascular and meningeal innervation of the head. Nerve fibers have in the past been described upon the blood vessels everywhere except in the placenta and the brain (Stöhr, 1928). The vessels of the pia mater bear upon them bundles of nerves both myelinated and non-myelinated, and also a fine branching nerve network. In the last two years, by certain technical modifications, we have been able to demonstrate that these nerve fibers are continued into the brain substance upon the intracerebral blood vessels. Nerve endings are found in the media of these vessels resembling small motor end plates. In the adventitia of the pial vessels, on the other hand, are found large nerve endings like Pacinian corpuscles (Fig. 2). These latter endings are evidently sensory although not necessarily concerned with pain sensation.

The source of the pial and intracerebral vascular nerves is firstly, from the sympathetic nervous system along the carotid arteries to the circle of Willis. Secondly, sympathetic nerves enter the skull along the vertebral arteries from the stellate ganglion complex. But there is a further source for these lepto-

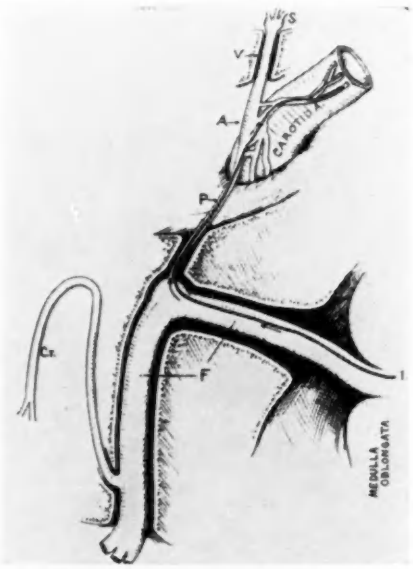

FIG. 3—Outline of vaso-dilator nerve pathway from medulla along facial nerve *F* and greater superficial petrosal nerve *P*, into the carotid plexus at the anastomosis *A*.

meningeal nerves. Dr. Chorobski, working in our laboratories here in Montreal, has described a small nerve which accompanies the seventh cranial nerve and leaves it to join the middle cerebral artery via the greater superficial petrosal nerve (Fig. 3). This may be called the vasodilator or parasympathetic supply to the brain.

Stimulation of the cerebral sympathetic nerves causes measurable constriction of the pial blood vessels (Forbes and Wolff, 1928), and stimulation of the cerebral parasympathetic nerve produces ipsilateral vasodilatation of those vessels (Chorobski and Penfield, 1932; Cobb and Finesinger, 1932). Thus both anatomically and physiologically the vessels of leptomeninges and brain have a double control similar to that elsewhere in the body.

There is a close relationship between this innervation and that of the naso-pharynx. Autonomic efferent fibers so far described in the facial nerve leave the medula oblongata with the roots of that nerve to be distributed peripherally by two branches, the greater superficial petrosal and the vidian nerves.

Vasodilator fibers pass through the chorda tympani to the blood vessels of the submaxillary and sublingual glands and of the anterior two-thirds of the tongue (Fig. 4). Similarly vaso-

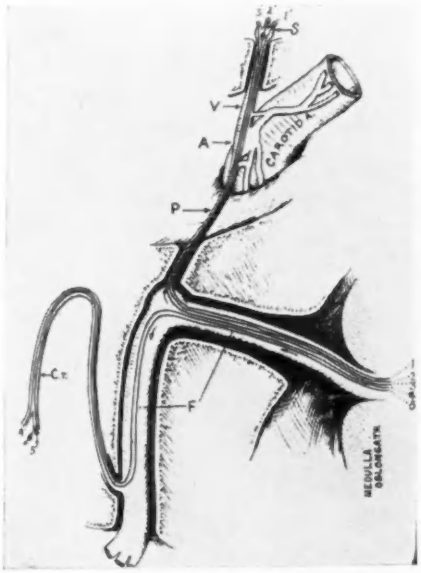

FIG. 4—Pathway for vasodilator impulses from the facial nerve through the chorda tympani, *CT*, and the greater superficial petrosal *P*, and vidian *V*, nerves.

dilator nerves pass from the facial through the greater superficial petrosal nerve which becomes vidian nerve to supply dilator fibers to the mucous membrane of the nose, upper pharynx, soft palate, roof of mouth and upper lip. These fibers have been shown to be interrupted in the sphenopalatine ganglion. Vasoconstriction in these structures is produced by stimulation of the cervical sympathetic.

Therefore, it is seen that, contrary to earlier teaching, vasoconstriction in the vessels of the pia mater is produced by stimulation of the superior sympathetic ganglion just as it is in the naso-pharynx and tongue. Vasodilation is produced by stimulation of an autonomic tract that passes by means of the facial nerve and the greater superficial petrosal to the pial vessels just as the dilator tract passes through facial, and either chorda or greater superficial petrosal to tongue and naso-pharynx.

Under pathological conditions I can testify to the fact that pial vessels are capable of complete spasm. On the brain of an epileptic patient after an epileptic seizure, I have more than once seen pial arteries develop points of complete constriction as though a thread had been tied about the vessel. When two such constrictions develop in one artery a segment of the vessel is converted into a sausage with pulsation on both sides but none in the middle. Under the same conditions I have seen circumscribed areas of anemia appear. In other cases a gyrus has blushed suddenly and then become pale. These changes are only possible if there is local control of the arteriolar or capillary bed.

To describe nerve fibers and even nerve endings upon the envelopes and vessels of the brain is one thing. To say that these nerves convey pain is quite another. Local anesthesia and *entré* to the cranial cavity gives the neurosurgeon an opportunity for unrivalled observation. My conclusions from such direct study is that the dura mater is of itself insensitive.

On the other hand, the middle meningeal artery and its branches give rise to pain when pressed upon. This pain is referred by the patient to his temple. In addition to sympathetic nerves this portion of the dura is supplied by the second and third divisions of the trigeminal. Histologically, there are numerous medullated and non-medullated nerve fibers upon the meningeal artery and its branches (Figs. 5 and 6).

FIG. 5—Nerves in dura mater near meningeal artery; note branching. Macacus Rhesus. Gros-Bielschowsky silver stain modified.

FIG. 6—Middle meningeal artery with accompanying nerve lying on adventitia. The intima of the artery is stained but not the wall. Note branches passing off on arterial branch. Macacus Rhesus. Technique as in Fig. 5.

The longitudinal sinus is very sensitive to pressure. The pain is referred by the patient to the top of the head. On one occasion it was referred to the face. The tentorium and particularly the dural sinuses within the layers of tentorium are very sensitive to pressure. The tentorium forms an incomplete diaphragm which extends across the cranial chamber. If pressed upon at any point it exerts a pull upon the exquisitely-sensitive sinuses, a pressure which must often be exerted no doubt by alterations of intracranial pressure. This pain is described by the patient as a pressure behind the ipsilateral eye. The tentorium is innervated by the first division of the fifth nerve.

Histologically, there are scattered nerves running about the sinuses (Fig. 7). Electrical stimulation or pressure produces the pain mentioned above. Even application of heat to them produces a sensation of pain, not of heat.

The leptomeninges are insensitive to cutting or pressure, but the passage of a bubble of air through them can be felt as a pain, sometimes sharp, which is localized accurately to the side of the head involved. The leptomeninges in addition to the

FIG. 7—Nerve fibers in wall of longitudinal sinus. Macacus Rhesus.

sympathetic and parasympathetic fibers described above are innervated by direct twigs from almost all of the cranial nerves.

Migraine, particularly unilateral migraine, gives many clinical evidences of associated vasomotor disturbance. There are also disturbances of the nervous system such as hemianopsia, ophthalmoplegia, numbness, paraesthesias and paralyses which only clear up sometimes after days and weeks. Such paralyses, like those which sometimes follow epilepsy, are probably the result of rather long-continued vasoconstriction. That does not mean necessarily that either condition is due to vasoconstriction. The vasoconstriction may be only a secondary phenomenon associated with epilepsy and with migraine. But it suggests that vasomotor change is important in their production.

Migraine, like angina pectoris, may have a number of different exciting causes. It may be precipitated by food idiosyncrasy, fatigue, constipation and the menses, but there is a constant underlying neurological mechanism which, in my opinion, probably involves an axone reflex in the sympathetic nervous system and afferent somatic nerves.

Presentation of Cases

A woman (G.M.) was referred to me by Dr. Robert Brink of Courtland, New York, thirty-eight years of age. She had complained of headache from the age of eight. The pain had always been situated in the right frontal region radiating down into the right shoulder. The attacks were preceded by a pallor of the face and a sensation of fullness in the nose. During attacks her right eye watered. The attacks recurred about every twelve days and had become unbearable even with very large doses of morphine. She was free from the headaches only during her two pregnancies.

As life hardly seemed worth living to her we decided to attack the problem radically until relief was obtained. Injections of the sphenopalatine ganglion by Dr. McNally gave temporary and partial relief only. Consequently, in September, 1929, the superior sympathetic ganglion was removed and the carotid decorticated. The upper pole of the stellate ganglion was then excised and the plexus upon the vertebral artery removed. This procedure was followed by the constant neuritic type of pain

which sometimes follows sympathectomy (Reid), and she complained of pain in the parotid gland on eating sour things, which may also be a sequel to sympathectomy. Further, she continued to have her headaches at regular intervals but the pain in the shoulder and neck had disappeared and now was only present in the right side of the head and face.

Three months later, in December, 1929, I injected novocaine into the Gasserian ganglion through the third division during a headache. The jaw became numb first, no change in headache; the anesthesia spread into the second division, still no change; lastly the forehead became numb and the patient remarked immediately, "I have nothing to feel the pain with." I therefore carried out a subtotal rhizotomy of the fifth nerve. But instead of cutting only the lateral posterior root fibers, as in Frazier's method, I reversed the procedure, thus rendering only the upper half of the face numb and sparing the tongue and lower jaw as well as the motor root (Fig. 8).

In the three years which have elapsed following her operation she has had no recurrence of headache. For a time she

FIG. 8—*Case G.M.* Complete loss of sensation in forehead above dotted line. Partial loss between dotted line and line to corner of mouth. Normal sensation below this line. Following reversed subtotal trigeminal rhizotomy.

felt periodically as though she should be having a headache but felt no pain. Even this recurring sensation has now disappeared. She has been, to quote from a recent, somewhat exultant letter, "about the healthiest person you ever saw."

Complete cranial sympathectomy relieved her of all radiation of the pain beyond the distribution of the trigeminal. How is it, then, that pain continued in this distribution? It is true that all sympathetic connections with the rest of the body had been removed. But we know that there are isolated ganglion cells within the leptomeninges and sometimes an accessory ganglion upon the carotid within the skull. These sympathetic axones have no outside connections but they may react within their own distribution and the pain be carried to consciousness along the route of the trigeminal. Or possibly the pain may have travelled directly along the fifth nerve without a sympathetic reflex.

Would it have been sufficient to cut the posterior roots of the fifth nerve? It seems to me unlikely, as axone reflexes through the sympathetic would still have reached the back of the head, neck and arm.

A second case illustrates this point. A woman (E.B.) of thirty years, referred to me by Dr. MacNaughton, came complaining of an atypical dull pain in the left temporal region, face and neck. This pain had begun three and one-half years before and had recently become constant, day and night. She suffered so greatly that she had prevailed upon a surgeon to make a trephine hole in the parietal bone at the point of greatest pain. This was done; of course, without relief. There was a Horner's syndrome of ptosis and small pupil on the left, and there was increased lacrimation in the left eye.

In March, 1930, the superior cervical sympathetic ganglion was removed and periarterial sympathectomy carried out on the carotid. The vertebral was not touched. The pain continued although it was now largely confined to the left temple. The trigeminal ganglion was injected and again the third and second divisions became anesthetic without relief. But as the anesthesia reached the forehead the pain disappeared. The anesthesia disappeared at the end of thirty minutes accompanied by the reappearance of the pain. Upper subtotal section of the sensory root was, therefore, carried out as in the other case (Fig. 9).

FIG. 9—*Case E.B.* **Complete loss of sensation in forehead.** Slight quantitative decrease in sensation within dotted line.

In the first twenty-two months since operation she was relieved of the pain of which she complained, has returned to her work and is extremely grateful. She did, however, continue to have a dull pain similar to the old one, though very much less intense. It was located chiefly in the posterior part of the left side of the head and the neck. Had the vertebral sympathetic been removed at the time of the first operation it is my belief that she would now be free from pain here, for the vertebral is able to spread the pain in the sense described above, beyond the territory of the fifth nerve.

Dandy has recently reported the cure of two cases of migraine by unilateral removal of superior cervical and stellate ganglia.* At the time of his report the cases were followed only over a period of two and six months, respectively.

* It has not seemed necessary to me to remove the stellate ganglion if all connection with the head be interrupted by excising the superior sympathetic ganglion from the carotid and the saddle-shaped ganglionic extension upon the vertebral artery. This latter artery cannot be reached from behind by the Adson approach used by Dandy.

So far as my experience goes this procedure has proven insufficient as indicated by the following case. In 1926, I carried out a unilateral sympathectomy, sparing the stellate ganglion proper, but removing the ganglion upon the vertebral artery and cutting the vertebral plexus as well as the superior sympathetic ganglion, as described in the first case above. The patient was a man of forty-eight years who had suffered for twenty-four years with recurring hemicrania associated with tachycardia, fatigue and lacrimation. This operation was followed by no improvement. Subsequent alcohol injection of the second division of the trigeminal then gave very considerable but not complete relief. The patient refused to carry on with the trigeminal rhizotomy so that the first case in which the whole procedure was tried was G.M., cited above.

In summary, unilateral migraine can be cured by radical neurectomy. The neurological mechanism involved may perhaps be similar to that which I believe obtains in angina and probably other forms of referred pain; i.e., an autonomic nerve being stimulated by some focal irritation serves to reflect the stimulus to the periphery at a distance by means of an axone reflex. The pain sensation is then carried to consciousness along the cerebrospinal sensory nerve, which innervates the peripheral part in question.

It has been pointed out that the innervation of the nasopharynx is quite similar to that of the meninges and it is quite likely that the hypothesis outlined above might be applied to intractable pain arising in this territory. Last winter I was called upon to address the Boston Orthopedic Society on the subject of referred pain. A short time later Dr. William Rogers, secretary of that organization, wrote me that he had applied the axone reflex hypothesis to two cases of chronic sacro-iliac pain. He had injected novocaine superficially into the skin where the pain seemed to be felt and the pain had been relieved at once.

So it is by analogy that we may receive most help from the work that is being carried on outside our own specialty. We in neurology are so deeply in debt to our ophthalmological and otolaryngological confrères for constant help and stimulus that it would be a great source of satisfaction to be able to reciprocate, and there is one interest common to all medical men of whatever type, i.e., the problem of the relief of intractable pain.

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IV. DISEASES OF THE EYES, EARS, NOSE AND THROAT FROM THE METABOLIC POINT OF VIEW

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I need hardly dilate upon my limitations in the fields of ophthalmology and otolaryngology; and I may tell you it was not without some diffidence that I accepted the invitation to address this Academy. My knowledge of diseases of the eyes, ears, nose and throat is limited to that which one cannot help but gain from daily association with those engaged in this work in hospital practice, and it is not far from that minimum essential in the ordinary practice of medicine. One engaged in such practice, and especially in the teaching of medical students, should, for ordinary physical examination, recognize the more common eye lesions; know how to differentiate a normal from an abnormal fundus and how to interpret the more common reports of the ophthalmologist on acuity and fields of vision. He should recognize the more common lesions of the ear and be familiar with the common bedside tests of hearing and vertigo. He should also know what the vocal cords look like normally, and be able to recognize an abnormal throat. The little additional knowledge which I may have gained came from requests for special investigation of the metabolism of patients and from familiarity with the necessary literature for interpretation of experimental data. These experiences have led me to formulate certain views as to the application of laboratory methods in ophthalmology and otolaryngology in diagnosis and treatment and the possible uses to which the laboratory may be put in the advancement of further knowledge in these special branches of medicine.

Though diseases of the eyes, ears, nose and throat are not entirely unrelated, these organs represent four different parts of the human anatomy and the field in each case is a wide one. In the time allotted to me, my remarks must, therefore, be rather fragmentary. I shall attempt to outline briefly the recent advances made in the study of metabolism in medicine in general and show how the ophthalmologist and otolaryngologist may take advantage of presently-available facilities.

The physician of the past felt, because of the limited facilities then available, had to feel, that he performed his duty largely when he succeeded in giving his patient's disease a name — when he succeeded in making a diagnosis. Correct diagnosis is obviously important at all times, but correct diagnosis alone does not necessarily afford correct treatment. Proper treatment depends upon proper appreciation of underlying pathology and much of this is now made possible by the application of metabolic studies. In the past, with a knowledge of anatomical pathology alone, we had to be satisfied with the solution of the question — What is abnormal? We now seek the solutions to the questions — Why is it abnormal? What is the mechanism of the production of a given clinical phenomenon and how may it be modified? The solutions to these questions lie largely in laboratory medicine — in the study of metabolism.

Every known phenomenon in this universe resolves itself into a chemical change, a physical change, or both. As this applies to the universe in general, it applies to human beings. Every phenomenon in health and in disease has as its basis a chemical change, a physical change, or both, and by the study of metabolism we mean the study of the chemical and physical changes which occur in the living cell.

It is hardly necessary to observe that such studies are now of more than academic interest. We meet with examples daily where, in spite of a careful clinical examination, diagnosis and recognition of the stage or the extent of a condition are not possible without recourse to laboratory methods. This applies to medicine in general and diseases of the eyes, ears, nose and throat afford no exception. On a former occasion I referred to the use of the laboratory in medicine and then stated that the physician may, if he wishes, continue the practice as in the past and be satisfied with clinical phenomenon alone. The oph-

thalmologist or otolaryngologist may, if he wishes, still confine his observations to a few square centimetres of mucous membrane; but if the results of such practice happen to be satisfactory, he should in the interpretation of his results, be mindful of the frequent and kind assistance of *vis medicatrix naturae*. Intellectual honesty made Ambrose Paré say, "I dressed him — God healed him." However, as I stated on a more recent occasion, it is no more our prerogative to do as we may wish. Those entrusted to our care are entitled to accurate diagnosis and proper treatment wherever possible; and the time is probably not very distant when the capacity of the physician to make use of the newer diagnostic facilities will determine his status in his profession.

What may be accomplished with the aid of the laboratory has been repeatedly demonstrated in medicine in general. Though the study of metabolism in disease is comparatively young, its accomplishments have been many and afford a striking example of what medicine owes to the exact sciences. We owe much to the physicist; studies of radioactivity have led to our use of X-rays and radium; to thermodynamics we owe the estimation of basal metabolism; to optics we owe the laryngoscope and the ophthalmoscope; to hydrostatics and hydrodynamics we owe determination of blood pressure, and because of the galvanometer we have the electrocardiograph. Chemistry has made possible the investigation of the composition of blood, urine and other body fluids and this has led to the development of the variety of tests of kidney and liver function and the present-day methods of diagnosis and treatment of diabetes and other disorders. Nor has the mathematician failed to play his part; to quote from a former address, mathematics are employed constantly in the co-ordination of the experimental results; the efficiency equation finds its applications in the study of hæmolysis, precipitation phenomena and the dissociation of hæmoglobin; the differential and integral calculus and trigometrical ratios are used in animal mechanics (muscular action); there are the numerous physiological applications of thermodynamic equations and the pathological chemist makes use of the Law of Organic Growth; medical statisticians apply the Laws of Probability and nomographic charts are used daily in the calculation of basal metabolic rates.

What can the laboratory do for those engaged in ophthalmology and otolaryngology? As in medicine in general, we may, for practical purposes, divide all diseases into three groups, namely, (a) those which we do not recognize as yet, (b) those which we recognize, but do not know how to treat, and (c) those which we may both recognize and treat successfully.

To deal with the first group would be time-consuming and largely a matter of speculation. I venture, however, to suggest that when we know more of functional pathology, a number of meaningless diagnoses will disappear. I always suggest to students, when their teacher in neurology uses the term "idiopathic epilepsy"; when the urologist speaks of essential hæmaturia, or the internist makes a diagnosis of neurasthenia, to ask the teacher what he means.

In the second group of conditions, our accomplishments end with diagnosis. Ochronosis — an inborn error of metabolism — may be cited as an example. Blueness of the ears and nose due to deep (cartilage) pigmentation and triangular brown patches of the sclerotics due to superficial pigmentation are some of the characteristics of this condition which are of diagnostic value. We may recognize inborn errors of metabolism, but do not know how to treat them. Fortunately, these conditions are rare and, in most instances, harmless, though harmlessness is not an essential attribute; there are, for example, the destructive lesions of the eye which lead to blindness in congenital hæmatoporphyrinuria; the defective vision and its sequelæ in albinism, and the tendency to calculi formation in cystinuria.

The majority of diseases, fortunately, belong to the last group, namely, those which may not only be diagnosed but also treated and it is here that the laboratory is found most useful. Lesions resulting from poisons may be cited as examples.

Ophthalmology and otolaryngology have their incomplete diagnosis as well as medicine in general. A diagnosis of "retinitis" is as incomplete as that of aortic regurgitation or auricular fibrillation. Etiology is one of the additional essentials for completeness and here the laboratory has its use. Conditions met with in industrial medicine may be cited, and I now quote from our own experiences. Not every person suffering from methyl alcohol poisoning has accidentally taken the methyl for the ethyl compound, nor does every individual poisoned with methyl al-

cohol develop sudden blindness with vomiting and abdominal pain. With the use of this alcohol in industry, the saturation of the body tissues from inhalation of the vapor may be insidious and only chemical examination of the urine may explain the true cause of disturbance of vision or other obscure nervous symptoms. Chemical examination of the urine, and again I quote from our own experiences, may reveal a retinitis due to lead poisoning, though there may be nothing suggestive in the clinical history, little or no pallor, no colic nor constipation and no albuminuria; and in the blood there may be no evidence of basophilic degeneration of the red cells. Chemical examination of the urine may also at times explain lenticular opacities in those exposed to naphthalene; and diminished central acuity and contraction of the visual field may be due to long exposure to nitro-benzol. Here, also, there may be no clear history. The laboratory findings tell the story. In the last case I know of, shoe dye was the source of the poisoning.

Diabetes, perhaps, affords one of the best examples of the part the laboratory may play in diagnosis and treatment. Lesions of the eyes which may be attributed directly to diabetes and which may be relieved in many cases by proper treatment of this disease are seen more frequently in clinics for diabetes than in those for diseases of the eye. Almost every other diabetic has some eye lesion. Of one thousand consecutive examinations by our ophthalmologist, Dr. S. H. McKee, abnormalities were found in 433 cases — an incidence of 43.3 per cent. This emphasizes the importance of routine urinalysis — a practice which, may I suggest, was not too common in the past.

From seeing many diabetics daily throughout the year, one cannot help but be impressed with certain phenomena as they are repeatedly observed and I have learned the wisdom of the following rules:

Cataract is not always an expression of old age. It may be the first indication of diabetes and with proper treatment of the latter, the eye condition may improve markedly or disappear entirely.

Alternating far- and near-sightedness should always make one suspicious of diabetes. Because of our routine examination of every patient on admission to our clinic, we have found this to be a very common phenomenon. The history is characteristic.

The patient consults a physician and is told he is suffering from far-sightedness, for which glasses are prescribed. The condition disappears, but shortly afterwards the patient again complains of faulty vision and consults another physician who tells him he is suffering from near-sightedness, for which glasses are again prescribed. Such expense may be avoided by examination of the patient's urine and, when necessary, examination of the blood.

There is the peculiar condition of blindness due to sudden reduction of hyperglycæmia. Usually the onset is sudden and the duration is short. Of practical importance is the fact that the ophthalmoscopic picture is not unlike that of cataract and I have a record of such a diagnosis having been made by an expert ophthalmologist. The condition readily disappears when the carbohydrate metabolism is stabilized. One of our own patients suddenly complained of disturbance of vision and became completely blind within forty-eight hours. Within a week the eyesight was perfect.

Urinalysis and, at times, blood examination, is, in my opinion, essential for the differential diagnosis between albuminuric and diabetic retinitis. According to textbooks, the ophthalmoscopic picture alone tells the story; but I have the impression that such diagnosis without examination of the urine and at times examination of the blood is largely the result of the time-honored custom of accepting statements unquestionably on mere authority. Unless I interpret the data wrongly, the pathological basis of the lesions in both diseases is the same; reduction in size of vessels, undue tortuosity, irregular calibre of vessels, displacement of veins from their course by crossing arteries, thickening of perivascular sheaths, hæmorrhages and scarring at sites of old hæmorrhages are all merely manifestations of vascular degeneration and may occur regardless of the cause of the vascular disease — they are findings common to both albuminuric and diabetic retinitis. Differentiation between the two conditions is obviously important from the prognostic point of view. In albuminuric retinitis, the prognosis, as is well known, is extremely grave, but I know of a number of individuals who have had retinitis following diabetes for ten years or more and, who, as yet, are still in fairly good health.

I stressed the importance of urinalysis. It may, however, be observed that such examination alone does not always suf-

fice. In the early stages of diabetes, the urine may frequently contain no sugar at sometime during the twenty-four hours, because the blood sugar is normal or nearly so; and when the disease is advanced and accompanied by vascular complications, the urine may contain no sugar, in spite of a marked increase of blood sugar. Such individuals have what is known as a raised renal threshold for glucose. Sugar ordinarily appears in the urine when the concentration of sugar in the blood is approximately 0.18 per cent. Diabetes with raised renal threshold may have blood sugars as high as 0.3 or 0.4 or more per cent and no sugar in the urine. One should always suspect a raised renal threshold for sugar when there is evidence of (a) arteriosclerosis, (b) chronic nephritis, and (c) infection. Gross dietary indiscretions may also lead to a raised renal threshold in diabetes and insulin may, *per se*, be a cause, especially in young people and in individuals who require large amounts.

Blood sugar determination should be a routine in the preparation of patients for operation, regardless of the type of operation. Failure of a clean wound to heal may be due to early diabetes, at which stage there may be hyperglycæmia with no gylcosuria. Control of the hyperglycæmia in such cases hastens healing, for which with proper pre-operative and post-operative medical care, the post-operative course of the diabetic should differ in no way from that of the non-diabetic. This statement is based upon experiences in this hospital with 523 operations since the advent of insulin; and not an inappreciable number of these operations — forty-five — were for conditions in which this audience is interested. They included operations for cataract, ethmoidectomy, mastoidectomy, sub-mucous resections, tonsillectomy, etc.

When the individual is known to be a diabetic, a problem which not infrequently arises is whether to operate first and then control the diabetes, or control the diabetes and then operate. It is not always a simple matter to decide. The possible effects of controlling the diabetes prior to operation in infection were strikingly shown in the following case.

The patient was admitted with the usual features of an acute mastoiditis. Clinically, he was very ill, with high fever, etc., but he was also suffering from severe acidosis of diabetes. There appeared to be little choice as to priority in treatment.

He appeared to be suffering equally from the diabetes and the mastoiditis. The usual rule is to remove an infection as quickly as possible, because, with infection, the patient may not respond to insulin. Because of a number of considerations, I suggested to the surgeon that an attempt be made to bring the diabetes under control before operation. To summarize the results briefly, when the diabetes was brought under control, the patient recovered completely from the mastoiditis without operation.

As with all aids to diagnosis in medicine, the laboratory has its uses and abuses and, perhaps, nothing does more to retard the advancement of knowledge than failure to use common sense in the practical application of experimental data. Practices with infection of the ears and sinuses may perhaps be referred to here. No one today doubts the existence nor the importance of vitamines in food. Their absence gives rise to definitely pathological lesions. Night blindness and xerophthalmia are, for example, two results of deficiency of vitamine A and there is much to suspect that infection of the sinuses and middle ears is another result of such deficiency. It does not, however, follow, because exposure of albino rats to vitamine A-free rations led to bacterial invasion of the nasal cavities and middle ears that herein lies the explanation of all or, may I suggest, even an appreciable number of such cases. Proof of a causal relationship between vitamine A deficiency and sinus infection or middle ear disease in a given case should be based upon a very careful history with regard to the individual's dietary habits and, in the interpretation of the history, consideration should be given to the wide distribution of vitamine A, both in plant (vegetable and fruit) and animal life. A careful history will probably more often than not make the physician seek some cause other than deficiency of vitamines and avoid exposing patients to unnecessary changes of dietary habits.

One valuable result of the use of laboratory methods is that it discourages dogmatism in diagnosis. Experiences with gout may be cited as an example. Gout is frequently manifested by lesions in the eyes. It does not, however, necessarily follow, because the individual has a condition in the eye which may be found in gout, and because the patient also complains of joint pains, that the condition is due to gout. Some lesions may be characteristic of gout, while others are merely suggestive; but,

may I suggest, in very few instances can a diagnosis be made without laboratory findings. According to the literature, the evidence of causal relationship is frequently of the slenderest and, as Foster Moore aptly puts it, "it would often seem less retrograde to uncover one's ignorance under so discredited a term as idiopathic, than to apply a title which, too often, is but a placebo to the patient, and to the surgeon a self-deception." Whether an individual is, or is not, suffering from gout is readily determined by presently available methods. Unless the lesion is pathognomonic, it should not be attributed to gout without a careful study of the purin metabolism, which includes a carefully controlled diet and examination of the uric acid contents of the blood and urine.

I cited a number of examples of the practical application of chemistry and physics in diagnosis and treatment. Chemistry and physics, however, still have their uses in the further advancement of knowledge of disease. Experiences with sinus infection and hyperthyroidism may be cited here.

Though infection of a sinus or middle ear implies bacterial invasion, conditions other than the presence of bacteria are essential for the production of lesions, for these parts of the body differ in no way from infection elsewhere. The sequelæ of bacterial invasion depend, amongst other things, upon the condition of the medium invaded; and I have the impression that investigation of physical phenomena may be useful here. There is much to suggest that barometric pressure and relative humidity are not unimportant factors and much might be discovered in the study of ionisation of the air. It has, for example, been fairly well established that positively charged air raises blood pressure and is generally stimulating; whereas, negatively charged air lowers blood pressure and causes depression. Stimulation and depression are not inappreciable factors in the resistance of the individual to infection in general. A low barometric pressure and a relatively high humidity are conducive towards retention of water in the body. Experiences with many thousands of renal test meal and polyuria tests in the study of kidney function afford proof of this fact. There is much to suggest that water retention may be the basis of the sensitivity of "rheumatic" patients to weather changes and their ability to predict rainy weather; with water retention, the fluid contents of joints in-

crease and the increased tension causes the pain. This phenomenon of retention of water with low barometric pressure and a relatively high humidity may explain the engorgement of the mucous membranes and the discomfort so frequently seen with periodic attacks of sinusitis.

The problem of tonsillar infection and hyperthyroidism affords much material for study. The association of these two conditions appears to be more causal than accidental; it appears to be greater than can be accounted for by the laws of chance alone. As a possible explanation of this association, I have often thought of Gaskell's theory of the origin of vertebrates. Though this theory has not the endorsement of biologists in general now, it still has its exponents. In the evidence which Gaskell presented to support his theory, there is the belief of a common origin of the thyroid, thymus, pituitary, kidney and the uterus. That disease of one of these organs may affect the other, because of their common origin, is suggested from a variety of clinical experiences. There is, for example, the association of thyroid enlargement and uterine function — the enlargement of the thyroid gland in pregnancy and during menstruation; there is the association of acute nephritis and tonsillar infection, particularly in children; there is the influence of pituitary extract upon the kidneys with respect to water excretion; and the frequent finding of diseased tonsils in hyperthyroidism. Because of our thousands of basal metabolic rate determinations made yearly, I thought it might be of interest to determine whether there was any difference between individuals with infection of the tonsils and those without with respect to the basal metabolic rate. The number of variables and the difficulties which immediately enter into interpretation of such data are obvious. Some of these variables may be present and not recognized, while others may be recognized and difficult to control. For example, one cannot with certainty differentiate between normal and diseased tonsils without microscopic examination of tissue. Infection of the tonsils may be obvious, but the infection may be so well localized that there is little or no absorption of toxic material, with little or no effect upon the general metabolism. Failure to note improvement of many conditions for which the tonsils were removed during the world-wide epidemic of tonsillectomies may probably be explained by such harmless foci of infection. Our first step in

this investigation was, therefore, merely statistical. Patients, when coming to the hospital for basal metabolic rate determinations, were, as a routine, asked, by the technician, whether they had their tonsils removed, and, if so, when. Here, again, a number of difficulties entered into interpretation of results. Individuals may have had their tonsils removed, but incompletely, and, as is well known, this was not an uncommon practice about a decade ago, because of the surgical technique then commonly employed. For this reason, the patients were also asked *when* the tonsils were removed. On the other hand, it does not necessarily follow that all tonsils have been removed completely since improvement of surgical technique. Another difficulty is that though the tonsils may have been removed completely, there is no proof that there may not have been tonsillar tissue elsewhere. However, because of the fact that the information sought was very little time consuming, the investigation was continued and it is of interest to note that, statistically, at least, there appears to be a relationship between basal metabolism and presence, or absence, of tonsils; the average basal metabolism of individuals with tonsils was approximately five per cent greater than those in whom the tonsils were removed; and, from calculation of Probable Errors of Arithmetical Means, it would appear that the difference found was not due merely to the operation of the laws of chance.

Other possible applications of metabolic work in ophthalmology and otolaryngology might be cited, but time does not permit. My purpose in this address was to point out the general trend in the practice of medicine; to show the possible practical applications of laboratory methods in the diagnosis and treatment of diseases of the eyes, ears, nose and throat and the possible uses to which the laboratory may be put in the further advancement of knowledge. If my remarks appeared rather fragmentary, I should again like to observe that, though these organs are not entirely unrelated, they represent four different parts of the human anatomy and the field in each case is a wide one.

SUMMARY OF SYMPOSIUM

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IN SELECTING this symposium the program committee has shown very commendable discriminating judgment. Too often special societies present collective papers on different phases of some subject, but the topic is largely considered from the specialist's standpoint alone. In this instance collaboration of the departments of medicine, surgery and physiology has been obtained, and this has entailed the excellent presentation you have just heard. My rôle is to apply to our specialties the various pictures presented.

Few could have explained so lucidly the reason for such class association between clinical observations and the complete hæmatological reports. I use the word "complete" since many blood examinations are very fragmentary and serious decisions are often made from such rudimentary information. The close association we have with general medicine has been very clearly brought out by Dr. Pepper. The normal blood picture is a visionary affair unless we view it with some elasticity. The white cell count is only constant under basal conditions, but under alteration of physiological states changes rapidly ensue. The doctor has pointed out how easily the white cell count may change, due to muscular activity, time of day, and if food has not been taken. We will have advanced a good deal if we know more about the function of the leucocyte in health and disease. No examination of a patient showing evidence of epistaxis, anæmia, chronic glandular enlargement, pharyngeal ulceration, should be considered thorough until a complete and intelligent study of the blood picture has been made by one who is familiar with the refinements of hæmatological technique and interpretation.

Dr. Keith has presented a most enticing study from the ophthalmological standpoint. I am quite unfitted to discuss his viewpoint, but I can appreciate to the full the aptness of his

remarks and the beauty of his fundus illustrations. His paper will be most interesting to the ophthalmologists present and from them he will, I am sure, appreciate a constructive criticism.

Dr. Penfield has chosen "Migraine" as his subject, rather than the broad one laid down in the program. There is some value in this, as to cover all that should be discussed in "Neurology in Relation to Diseases of the Eye, Ear, Nose and Throat" would be beyond anyone's ability in the time allocated. Dr. Penfield has, however, made my task easy in that he has presented viewpoints on the treatment of migraine that he knows are beyond the ken of the otolaryngologist and ophthalmologist, almost solely in the sphere of the neurological surgeon.

Migraine has run the gamut of all varieties of treatment by practitioners. This in the main has been disappointing, since we do not really know what causes migraine, or why the pain comes in bouts and so suddenly ceases. We have been searching our therapeutic storehouses in the hope of finding some drug that we could depend upon to give prompt relief. Professor Rudolf speaks highly of the use of antipyrine, 10 grains at once; 5 grains in half an hour; repeating in another half hour until 20 grains are administered in one and a half hours.

All of the specialties have had their fling at the treatment of migraine, for as unilateral supraorbital pain is the outstanding symptom, it is quite natural that the rhinologist should see many whose nose and sinuses have come under suspicion. One must remember that unilateral frontal pain is very often associated with acute antrum suppuration, as well as acute frontal sinus disease. Many cases of true migraine have had nasal treatments and operations owing to pathological changes in the nose or sinus masking the true ailment. Probably I am not alone in having resected a supraorbital nerve for supraorbital pain not recognized as migraine.

The rhinologist is not alone in his mistakes about this disease. The ophthalmologist has made his errors in diagnosis, for how many people have had their eye-glasses altered time and again, the weakest cylinders changed even .12 D., or axes altered a few degrees, with the assurance that these slight changes will alter the patient's complexion of life. The use of prisms and muscle exercises have been disappointing; so, too, has operative work on the muscle.

The gastroenterologist, too, has come to the rescue. He will point out the well-known fact that some patients who eat ice cream too fast will experience a severe supraorbital pain, and from this he postulates the relationship between duodenal or other intestinal upset and the attacks of migraine. Sometimes it takes a long series of diets, fasting, stomach lavage or wearing a Sippy tube to cure the delusion.

The roentgenologist also has had his obsession. X-ray has shown a small space between the two clinoid processes with an apparent crowding of the pituitary body. X-ray treatments have ensued, but the result is the same. Only those with a large functional element in this matter are better, as in all cases when any new or novel treatment is administered.

Not to be outdone by the other subdivisions of medicine, the gynecologist has had his say. Slight variations from the normal or even definite pathological conditions of the uterus or ovaries, have had whatever attention seemed indicated. The occasional relief experienced has in the end justified the observation that it is unwise to generalize from the occasional. Yet one must remember that there is still some virtue in experimental treatment, and in this connection your attention is drawn to the paper of Blackie and Hossack in the Canadian Medical Association Journal of July, 1932, in which the use of an ovary-stimulating hormone of the placenta has been followed by very happy results. That migraine attacks occur most often in females about the time the ovaries are beginning to become active, and cease during pregnancy and at the menopause, gives the hypothesis presented a reasonable background, and the citation of twenty cases adds impressive clinical support to the authors' contention.

The metabolic studies now occupying such an important place in medicine have their special application to our other problems. It would have been difficult to select anyone better fitted for this task than Dr. Rabinowitch. His ability to dovetail clinical research and experimental physiology has been amply shown by his remarks. I am sure, had time permitted, he would have cured most of the dietetic fanatics of their obsessions.

OPHTHALMOLOGIC SESSIONS

RECENT DEVELOPMENTS IN TRACHOMA INVESTIGATIONS

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THE OBJECT of this paper is to set down recent results of investigations and opinions of competent research workers and clinicians in the field of trachoma. An attempt has been made to produce a correlated and harmonized statement on each important phase of the problem out of the vast material that has been pouring into print. The difficulty of doing this is apparent when one is confronted with several languages, with different expressions which mean the same thing, and with descriptions of various phases of the disease which must be put in their proper order.

It has frequently been said that the problem of attempting to determine the etiology of trachoma has been made exceedingly difficult by the confusion regarding the clinical picture. On the one hand the laboratory worker has wanted the clinician to tell him definitely what cases were trachomatous and what were not; on the other hand the clinician has desired the laboratory man first to furnish him the specific criteria for making a differential diagnosis. Fortunately those most familiar with the clinical aspects of trachoma are now practically agreed on the diagnosis except possibly of cases in the very earliest stage. The laboratory worker may at least be provided with positive cases.

I. CLINICAL ASPECTS:

Since the matter of making a diagnosis is most important it seems best to endeavor to clarify first the clinical picture of trachoma. The earliest manifestations of the disease have been studied recently with great care by a number of workers in both experimental and clinical cases. The biomicroscope has added greatly to the facility and thoroughness of these observations. In fact, this instrument is now considered of more value than

the microscope in making a diagnosis, particularly in the early stage which is the one that gives us the most trouble.

The earliest visible changes occur in the network of vessels in the upper tarsal conjunctiva. The larger vessels are congested, in consequence of which the delicate capillaries become so highly developed and swollen that the surface of the conjunctiva appears studded with numerous tiny red dots or punctations, here and there interspersed with whitish circular areas larger than the punctations. Wilson[1] refers to these first changes as the prototrachomatous stage of trachoma, a stage preceding Trachoma I of MacCallan.[2] Under the high magnification of the biomicroscope each red punctation is seen to be a veritable bouquet of capillaries situated just beneath the epithelium (Cuénod and Nataf[3]).

The pale circular spaces which, by the way, are considered the first manifestation of the trachomatous follicles, are generally located between bifurcations of blood vessels and are also more or less encircled by smaller vessels. To the naked eye these embryonic follicles appear first as tiny yellowish-white points on the tarsal conjunctiva as was pointed out by a British colleague of mine in China, Dr. E. J. Stuckey,[4] who for two years during the war was ophthalmologist and medical officer in charge of Chinese labor battalions in France. Stuckey contended that these points are absolutely diagnostic of beginning trachoma. But Cuénod and Nataf think that the changes that follow definitely establish the diagnosis.

Due to a loss in the normal transparency of the conjunctiva most of the larger vessels as well as the delicate capillaries soon become no longer visible. With the slit lamp the capillaries are seen to have been replaced by a marquetry of polygonal bright-red plaques or mosaics which are separated by clear linear spaces. The red stippling is caused by the presence of a terminal capillary tuft or bouquet in each plaque. The plaques represent the beginning of trachomatous papillæ. The individual papillæ steadily hypertrophy, push outward and eventually become closely packed together. On account of the lateral pressure of the papillæ upon each other and the increasing dominance of the hemispheric opalescent nodules or follicles which separate the plaques here and there, the papillæ generally remain very fine, often invisible to the naked eye. But they are unmistakably clear

with the biomicroscope, and also in microscopic sections. The papillæ gives to the tarsus a velvety appearance even in the presence of the follicles which are more in evidence at the upper tarsal border.

Synchronously with the changes occurring in the tarsal conjunctiva, the bulbar and the retrotarsal conjunctiva become more and more congested and somewhat edematous. In the retrotarsal area and the fornix the conjunctiva becomes so redundant that it is thrown into horizontal folds in which follicles soon appear. At this stage there usually is a watery - mucoid discharge. In acute cases the discharge is muco-purulent.

As the disease progresses from Trachoma I to Trachoma II according to MacCallan's classification, the follicles in the retrotarsal conjunctiva become larger and more translucent, and frequently rupture spontaneously, a phenomenon which is characteristic only of trachoma (Wilson[1]). The bulbar conjunctiva becomes more and more hyperemic. But most important is the subepithelial infiltration which involves the entire conjunctiva and gives it a cloudy lusterless appearance. Follicles also appear in the bulbar conjunctiva, especially in those areas adjacent to the upper and lower fornices, in the semilunar fold and at the limbus. Later the caruncle itself is often studded with follicles around its base. According to Pascheff[5] the follicles in both the palpebral and bulbar conjunctiva tend to become confluent. To this confluent follicular hyperplasia he has given the name "folliculoma."

Along with the formation of follicles in the retrotarsal conjunctiva there also occurs the development of papillary hypertrophy (MacCallan's Trachoma IIb) as was described in the tarsal conjunctiva. But the proportion of papillæ to follicles varies greatly in different cases, so much so that we have been taught to speak of follicular trachoma if the follicles greatly predominate, of papillary trachoma if the papillæ predominate, and of a "mixed type" if the two forms are both clearly interspersed in the conjunctiva. During these stages (IIa and IIb) when the symptoms are generally less acute than they are in the initial stage, the discharge is mucoid and sticky and produces a dried yellowish film on the margins of the lids (Wilson[1]). It is quite generally agreed that this is the most infective period of the disease.

The formation of scar tissue is the next important phenomenon in trachoma. Cicatrization may actually appear in the initial stage or it may not appear until a later stage (Trachoma III). Trachoma is a self-limiting disease only because of the transformation of some of the subepithelial structures into connective tissue. In some cases this occurs early and quickly; in others more slowly, eventually becoming complete (Trachoma IV) if the individual lives long enough. Braun[6] states that trachoma may heal spontaneously at any stage, but rarely without conjunctival scarring.

In their biomicroscopic studies, Cuénod and Nataf[3] found as the first evidence of scar tissue a white horizontal streak across the tarsus devoid of follicles and papillæ. This has long been known as the cicatricial line or zone of Arlt,[7] due to its first being demonstrated by that ophthalmologist. It should be mentioned that this corresponds to the zone where the capillaries of the ascending and descending perforating vessels of the tarsus meet and anastomose. There next appear tiny irregularly star-shaped cicatrices in the tarsus connected by fine cicatricial lines or prolongations from the stars, each of which occupies the former site of a follicle. The follicles had previously either ruptured spontaneously and the cellular elements evacuated, or the cellular elements have become necrotic and partially absorbed. In either case the follicles are replaced by connective tissue fibers which had already invaded the follicles in an earlier stage.

Between the cicatricial areas of the tarsal conjunctiva there persist for some time granular islets which, however, gradually diminish in size and number as the scar tissue formation increases. These islets are made up chiefly of dark red vascularized papillæ with here and there a degenerating follicle. In the final stage the granular islets have entirely disappeared, the surface of the conjunctiva is quite smooth and whiter than normal and also devoid of vessels except for an occasional capillary.

The same cicatricial changes, preceded by evacuation or degeneration of follicles, occur in the conjunctiva of the fornix but on account of the looser structure of the tissue and the better blood supply, the formation of connective tissue is not so apparent.

In serious cases the cicatricial changes in the conjunctiva of both upper and lower lids may be very dense and result in

serious distortion of the lids, the upper generally being more seriously affected, with such complications as trichiasis, entropion and symblepharon (Trachoma IV). Wilson[1] thinks that fine scarring of the conjunctiva is in itself not pathognomonic of trachoma, for similar changes are seen following severe acute purulent ophthalmias. Fine scar tissue has frequently been reported following inclusion blennorrhea. Fuchs[8] states that one often finds scar tissue in the conjunctiva after recovery from a severe gonorrheal conjunctivitis. However, Cuénod and Nataf[3] declare that the tiny star-shaped cicatrices which may be seen with the biomicroscope in the tarsus some time before they become visible to the naked eye are absolutely pathognomonic of trachoma. With regard to the significance of extensive scar tissue formation there has never been any question. To establish the earliest criteria of diagnosis is our object here.

It is especially gratifying that regarding the question of pannus those most familiar with trachoma are now in almost universal accord. The corneal changes in trachoma are no longer looked upon as due to mechanical irritation but as an essential part of the disease. Here again the biomicroscope is of great value in making examinations.

The investigations of Vogt,[9] Gallemærts[10] and others with the slit lamp have established the existence in normal eyes of a transitional zone at the limbus containing (1) a highly developed lymphatic network with radial striæ which are pigmented, palisaded and anastomosed, and (2) a network of capillaries which appear surrounded by a double-walled lymphatic sheath and which generally anastomose. Occasionally one observes in normal eyes a tiny capillary penetrating a bit beyond the palisaded zone of the limbus into the transparent cornea, but such an isolated adventurous capillary is an exception and is in no sense analogous to the pannus of trachoma, which consists of superficial new vascular formation associated with subepithelial infiltration.

Cuénod and Nataf[11] have demonstrated that, at quite an early hour in the initial stage of trachoma, the first indications of pannus occur at the limbus of the upper corneal segment. Even before there is any involvement of the cornea visible to the naked eye, the biomicroscope reveals a number of tiny capillaries that have adventured beyond the limbal zone into the

transparent cornea. The high magnification also shows a fine infiltration not only between and around the capillaries but also in advance of them. That is, the lymph-cellular elements precede the capillaries, seeming to form for them jackets into which the capillaries enter and grow. Morax[12] is one of several others who also believe that the capillaries follow the cellular infiltration.

In addition to Cuénod and Nataf numerous investigators now aided by the slit lamp, are firmly convinced that pannus begins in the earliest stage of trachoma, probably even synchronously with the first signs in the palpebral conjunctiva. Wilson,[1] referring to trachoma in Egypt, declares that sooner or later all cases show the formation of new vessels in the cornea. In fact, he has never seen a case that did not. His statement quite confirms the opinion that I have maintained for years — an opinion based upon observations of trachoma in four continents: If a case did not show pannus within four to eight weeks, then it would never show any and was not a case of trachoma. I have never seen that principle fail.

There seems to be no definite relationship between the severity of the conjunctival involvement and that of the cornea. The conjunctiva may be markedly affected and the pannus be comparatively slight, or the conjunctiva may be only moderately involved and the corneal changes extensive. I recall two cases which were shown to me in Peking by my Chinese colleague and assistant, Doctor H. T. Pi, in which there were no conjunctival symptoms at all, but in which pannus was visible with oblique illumination. We admittedly were puzzled and decided to watch the cases without giving treatment. We were able to follow one of them long enough to see the conjunctiva become definitely affected with trachoma. Based upon the consensus of opinion, pannus is therefore recognized as absolutely pathognomonic of trachoma and always appears either synchronously with, or during, a period of not more than two months from the onset of the initial signs in the conjunctiva.

During the development of pannus in the cornea, cellular changes are occurring at the limbus leading to the formation of follicles. Later on these follicles may rupture and cicatrize to form ocelles (Bonnet[13]) or the marginal pits of Herbert (MacCallan[14]). The presence of these pits is considered a definite pathognomonic sign of trachoma. However, the formation of

follicles does not end at the limbus, but actually extends into the cornea (Pascheff,[15] Hiwatari,[16] Meyerhof[17]) where small rudimentary follicles or cellular concentrations appear, usually situated at the bifurcation of a large capillary. Pascheff calls these corneal concentrations true trachomatous follicles since their contents are identical with the elements of a trachomatous follicle of the conjunctiva. A number of these punctations may often be seen with the naked eye, generally just in advance of the terminal capillaries. Moretti[18] mentions small gray superficial dots in the cornea between the loops of vessels and at the ends of the vessels. In describing the corneal involvement in trachoma the writer[19] stated several years ago that along with beginning pannus "tiny infiltrations are formed beneath the corneal epithelium which may be referred to as a superficial punctate keratitis."

Superficial erosions of the cornea frequently occur. These generally appear as tiny ulcers where the follicles or superficial punctate infiltrations have emptied. The ulcers almost always heal quickly but leave permanent facets in the cornea. The facets in the cornea are the homologues of the star-shaped cicatrices in the tarsal conjunctiva (Cuénod and Nataf[3]).

As the disease progresses the vascularization and infiltration of the cornea followed by connective tissue formation increase. Whereas in the beginning only the superficial layers of the cornea are involved, eventually the process may extend deeper than the middle layers of the cornea. The corneal condition, instead of being a pannus tenuis, finally becomes a pannus crassus.

II. Histopathological Aspects of Trachoma:

More work has probably been done on the histopathology of trachoma than on any other phase of the disease. But even here our knowledge is not static as is evidenced by the recent contributions of several investigators.

It is very generally accepted that associated with congestion of the blood vessels in the palpebral and bulbar conjunctiva there always occurs a pronounced subepithelial infiltration of lymphocytes in the very beginning of trachoma (Kunz[20]). Also that its lymphocytic character changes more and more into an infiltration of plasma cells (Birch-Hirschfeld[21]) which likewise are present in the initial stage but which apparently develop more slowly

than the lymphocytes. Plasma cells proliferate chiefly along the blood vessels, possibly because they are the offspring of the vascular adventitia. However, Maximow[22] contends that they originate from small- and medium-sized lymphocytes. The fundamental constituents of the papillæ in trachoma are plasma cells, while the follicles possess typical germinative centers composed of monocytes, chiefly endothelioid cells, surrounded by a zone of lymphocytes. As the disease progresses plasma cells infiltrate the deeper tissues, especially the tarsus where in later stages they may be found occupying the sites of deteriorated meibomian and Krause's glands. Evidence of a general tarsitis is considered by some authorities as a definite diagnostic sign of trachoma. Ichikawa[23] maintains that in evaluating animal experiments, particular attention should be paid to the histologic changes in the bulbar conjunctiva, inasmuch as he considers the appearance of subepithelial infiltration of the bulbar conjunctiva which is rich in plasma cells, the only pathognomonic symptom of trachoma.

In occasional cases of trachoma, the proliferation of plasma cells is so strong that they even replace the lymphocytes of the follicles, or they themselves may produce follicles *de novo*. This probably corresponds to what Pascheff[15] has called plasmacellular conjunctivitis. Sometimes the follicles continue to enlarge and coalesce to form tumorlike structures which are known as plasmoma. These tumor masses as well as the more generalized plasma-cell infiltrations may undergo hyaline and amyloid changes. Recent authors including Soudakoff,[24] Pascheff,[15] and Papolczy,[25] on the subject of plasmoma are inclined to the hypothesis that all plasmomas are trachomatous in origin on the basis that nearly all of their cases also had trachoma. James[26] recently reported a case of plasmoma from our clinic which also had trachoma. I have seen ten cases of plasmoma and although the majority of them had definite signs of trachoma, I cannot subscribe to the above hypothesis; first, because the disease is too rare and trachoma too common to postulate such a relationship; second, because in two of my worst cases there was no evidence of trachoma although the tumor in lobulated straw-colored masses involved the fornices, the entire bulbar conjunctiva, and in pendulous fashion partly covered the cornea and protruded from the lids; third, because of the histologic finding in one

trachoma-free case of a calcified cyst which may have been the degenerated remains of an animal parasite of some kind. I should like to suggest, therefore, the use of the terms "primary plasmoma" when no complicating disease of the conjunctiva is evident, and "secondary plasmoma" when associated with such a disease as trachoma.

Associated with the formation of papillæ packed with plasma cells there is also an increase in the number of layers of epithelial cells. At the same time, due to the pushing out of the papillæ with their terminal capillary buds, there is an apparent downgrowth of epithelium between the papillæ. According to Wilson[1] these epithelial crypts frequently become blocked off and then one sees under the microscope epithelial pseudo-cysts filled with leucocytes and epithelial débris, which later degenerate with the formation of calcereous deposits. In the same way retention cysts in Krause's and the meibomian glands are formed.

According to v. Hippel[27] pannus is formed either in front of or behind Bowman's membrane, but the primary development of the corneal vessels on its posterior surface is the more frequent. Later this membrane becomes filled with perforations, giving it a moth-eaten appearance, and finally may disappear entirely.

The cause of scar tissue formation in trachoma has puzzled pathologists for many years. Its visible presence in the conjunctiva has long been considered one of the diagnostic signs of the disease. There have been many theories regarding the causes of its formation, but none has been proved thus far. I believe that Peters[28] in his latest work has advanced our knowledge on the subject somewhat. He has long advocated that the trachomatous changes in the conjunctiva consist in a deposit of lymphoid tissue which possesses a pronounced tendency to cicatrize and shrink.

Following a paper by Rössle and Yoshida[29] on the finding of a reticular tissue of lattice fibers in lymph glands both in normal and pathological conditions and the subsequent finding of similar fibers in numerous organs by other investigators, Peters demonstrated their presence both in acute and chronic trachoma. He also recognized a transition of these fibers in some places into collagenous fibers. From this he concludes that lattice fibers are a pre-stage of collagenous fibers. In the chronic cases he found a strong network of lattice fibers in the region

of the upper retrotarsal fold. He found them especially numerous around the follicles but much less frequently within the follicle itself. Most important of all he found them matted between the follicles and the epithelium as an additional layer.

Preparations of sections from a typical cicatricial case of trachoma showed the lattice fibers no longer recognizable, and the whole tarsal structure, very poor in cells, consisted of collagenous fibers. Peters concludes, therefore, that the lattice fibers in the conjunctiva may at least introduce the process of cicatrization, in the same way that they do in sclerotic processes of the lymph glands and in the liver. If Peters be correct it now remains to determine why the virus of trachoma has such a specific or selective action upon the lattice fibers, and also what happens to the lattice fibers in other diseases of the conjunctiva. This work has the promise of opening the way for a new method of differential diagnosis.

Two Russian investigators, Dwijkoff and Lewkoewa,[30] recently reported that they had demonstrated the presence of specific cells in trachomatous tissue by the application of certain pathological procedures and staining methods. However, Lamb working in our laboratory was unable to verify their findings.

There is no indisputable evidence that specific blood changes occur in trachomatous cases. Comparing the results of examinations of infected and of non-infected persons living in the same district, Marcus and Weiner[31] found no essential difference in the hemoglobin, erythrocytes, leucocytes, neutrophiles, eosinophiles and lymphocytes. A few authors think that their examinations have shown a moderate lymphocytosis but due to lack of a sufficient number of proper controls their conclusions are open to criticism.

III. The Follicle and Its Significance:

As one reads the recent literature he is impressed by the overwhelming belief in the dualistic as against the unitarian theory of trachoma. The unitarian theory holds that all follicle-producing affections of the conjunctiva have the same etiology. Perhaps a more consistent term that "unitarian" would be "monistic." For that matter the term "dualistic" does not so adequately express the opinion most generally accepted as does the word "pluralistic" which implies a theory that there are several follic-

ular diseases of the conjunctiva from an etiological standpoint. Blatt[32] considers the unitarian viewpoint, because of the complications of official regulations and measures, to be not only incorrect but dangerous, for if a patient with follicular granulations of the conjunctiva is interned in a trachoma hospital, he may contract real trachoma. He further says that unitarians overlook the fact that folliculosis is a benign condition which heals without scars and corneal involvement, while trachoma is a malign disease, the outstanding characteristics of which are scar formation and pannus.

Judging by the importance given to the conjunctival follicle in experimental trachoma by some recent investigators one would be inclined to believe that they considered the follicle specific of trachoma. It does not seem to be fully realized that the great majority of pathologists are opposed to this view. Pascheff[15] declares that the production of follicles does not mean that trachoma has been reproduced. Taborisky[33] states that it is an error to regard follicles as the *sine qua non* of trachoma diagnosis, since trachoma without follicles is not a great rarity even in adults, and often occurs in children. Kunz[20] says that the granule in the conjunctiva is not typical of trachoma, for the structures that develop in follicular catarrh and various chronic irritations do not in themselves show any morphologic deviation from the trachoma follicle which, like follicles of other affections, is only a form of reaction on the part of the conjunctiva to inflammatory irritation. In Birch-Hirschfeld's[21] opinion the follicle is not the essential (specific) factor for the course of the disease in trachoma. Rotth[34] states that the follicle does not signify trachoma for other irritations induce its appearance. He considers that the fully-developed follicle of trachoma differs clinically from other follicles but that in fresh cases there is no difference. Wilson[1] states that from a histological viewpoint there is very little to distinguish the early stages of trachoma from other follicular conditions of the conjunctiva. In trachoma he finds more evidence of a widespread inflammatory reaction resulting in a marked generalized subepithelial infiltration, but he considers "of much greater diagnostic importance the degenerative cicatricial and corneal changes which invariably follow in trachoma but which never occur in follicular conjunctivitis." Ditrói,[35] who is a proponent of the dualistic view and of the

unconditional specificity of trachoma, considers important from the standpoint of differential diagnosis the coppery-red color and general infiltration of the conjunctiva, and the bursting of follicles often followed by a new formation of follicles.

Morphologically speaking, there are two distinct types of folliculosis, the one, consisting of small pearly-looking follicles often arranged more or less in parallel rows on the palpebral conjunctiva; the other, characterized by massive, often confluent opalescent follicles generally restricted to the retrotarsal and bulbar conjunctiva but occasionally also involving the conjunctiva of the tarsus. It is this second type of folliculosis with the large soft follicle, particularly when associated with a mild inflammation, that is often mistaken for trachoma. Lindner[36] and Rieger[37] have adequately described this type which they consider an infectious process but which never leads to pannus and connective tissue formation, and tends to heal spontaneously in the course of several months to two or three years without sequelæ of any kind. As ophthalmologists we have all seen too many of these cases, often severe and extensive, and generally in children, entirely recover without treatment to believe that they are cases of trachoma.

This question of follicles is important because of its relationship to experimental trachoma in animals. Much has recently been written about spontaneous folliculosis in monkeys. Wilson[38] describes two types that he found in monkeys in the Zoological Gardens in Cairo, the first, consisting of small, superficial, hard, translucent, discrete follicles on the conjunctiva but not involving the tarsus; the second, a follicular conjunctivitis with large soft follicles, translucent and somewhat yellowish, associated with a subepithelial inflammation and a sticky mucoid discharge. He found the tarsal conjunctiva to be free. In early shipments of monkeys to our laboratory from New York we found so many of the animals with spontaneous folliculosis that we finally had to have them sorted before they were shipped either by one of us or by someone whom we could trust. For instance, Weiss examined a shipment of twenty rhesus monkeys at our laboratory on January 2, 1930, and found several with an extensive follicular and papillary hyperplasia involving the caruncle and the conjunctiva of the tarsus as well as the retrotarsal conjunc-

tiva, and associated with a slight muco-purulent discharge and some crusting at the lid margins.

Many other authors report the frequency of spontaneous follicvlosis in various species of monkeys and also in other animals. Nicólle and Lumbroso[39] found marked spontaneous granulosis in rabbits and on this account retracted the alleged successful transmission of trachoma to rabbits previously reported by Nicólle[40] and other coworkers.

In view of these follicular complications it is very difficult to evaluate the conjunctival lesions in experimental trachoma in animals, especially in the monkey. Without the association of pannus and scar tissue, the characteristic signs of human trachoma, it would seem unscientific to attach much importance to the follicle.

IV. Infectivity, Transmissibility and Epidemiology:

In spite of all the variations in the clinical picture, Heymann and Rohrschneider[41] emphasize that trachoma is a disease of distinctive character, whose infectivity has been unquestionably certified as a result of epidemiological experiences and experimental investigations. Noguchi[42] states that "the only reliable knowledge we possess regarding the inciting agent of trachoma is that it occurs in trachomatous lesions and is capable of being transmitted to certain species of animals."

There are many cases on record of accidental and experimental transmission of trachoma to human beings. One classic example should suffice. Professor Ernst Fuchs once told me that while one of his assistants was operating upon a trachomatous case some of the secretion was discharged into one of his eyes. The assistant ran into the adjoining room where Fuchs immediately treated him so vigorously and thoroughly with a solution of two-per cent silver nitrate that there followed an intense and distressing reaction. In spite of this, the assistant succumbed acutely to the disease in less than a week, and treatment was at once instituted. The other eye eventually became involved, pannus and scar tissue developed, and treatment was continued for more than two years before the disease became inactive. The term "inactive" is certainly the proper one to use rather than the word "cured" in cases of trachoma. I quite agree with Cohen[43] and Wilson[1] when they state that trachoma

is practically an incurable disease, and also with Davis's[44] declaration that "a quick cure of a conjunctival condition diagnosed and treated as trachoma is almost a guarantee of a mistaken diagnosis." Furthermore, the failure of the silver-nitrate solution to prevent such an infection as that related by Fuchs and confirmed by the experience of others (Lindner and Axenfeld) makes us feel that possibly the virus of trachoma is resistant to silver nitrate. Heymann and Rohrschneider[41] draw an analogy when they speak of the failure of Credé's method to prevent inclusion blennorrhea, while it is most effective against gonococci.

It is significant that in nearly all cases of accidental and experimental transmission of trachoma the disease begins with acute or subacute symptoms, whereas most of the textbook descriptions refer to it as an affection whose onset is insidious. Various explanations have been offered regarding this seeming disagreement. Some have suggested that the acute symptoms are always caused by a mixed infection, but this opinion now seems to be largely discredited. Some believe that the massive inoculation generally received in accidental and experimental cases as against the probably slight inoculation, often by dried and supposedly attenuated virus, that occurs in the so-called insidious cases, accounts for the different types of onset. Others believe that the presumably insidious cases begin more or less acutely but since the acute stage is almost always followed by a period of relative quiescence for several months before the symptoms slowly reappear, the relation between the acute beginning and the insidious return of the disease is not associated (Taborisky[33]). It would therefore be justifiable to conclude that although trachoma perhaps is an insidiously beginning disease in the majority of cases, there is such a thing as acute trachoma which develops without any evidence of a mixed infection with pathogenic bacteria (Blatt[45]).

The period of incubation of trachoma ranges from five to twelve days with the majority of cases showing the first signs in about a week. It should be added that the period of incubation is the same in inclusion blennorrhea, a disease whose infectivity has also been definitely established. In fact, it is probably highly contagious, while trachoma on the other hand is considered by most authorities to be only mildly contagious (Cohen[43]). Inclusion blennorrhea of the newborn has been experimentally

transmitted to adults by several investigators. The disease begins acutely and remains acute for a month or six weeks after which it slowly recedes over a period of several months to a year or more, finally healing without pannus and generally without scar tissue formation. According to Heymann and Rohrschneider,[41] Lindner, Siegrist, Wolfrum and others have reported the frequent finding of fine scarring in the conjunctiva of healed cases. Taborisky[33] says that the cicatrization is sometimes severe. Heymann and Rohrschneider[41] state that the inoculation of baboons with material from cases of inclusion blennorrhea is practically 100 per cent positive which is quite a contrast to the low percentage of positive inoculations with trachomatous material. All in all, therefore, it is difficult to subscribe to the theory that inclusion blennorrhea and trachoma are one and the same disease, or that inclusion blennorrhea is etiologically a genital trachoma as was suggested by Lindner[46] and the writer[47] several years ago. Although their beginnings may often be alike, they certainly terminate differently.

The question of monocular trachoma has brought forth a great deal of discussion, some of it quite recent. There are too many cases recorded by competent observers to deny the existence of such an affection. I have seen a number of such cases myself, the last one in our clinic in St. Louis about a month ago. In this young man who had been suffering with the disease for two years, one eye was seriously involved with a diffuse subepithelial infiltration, papillo-follicular hyperplasia, intense pannus and considerable scar tissue formation. He had photophobia and hemiptosis of this eye but no discharge. The other eye, which was examined minutely with the slit lamp, was absolutely normal. What is the explanation and what are the experimental data regarding such cases?

Several investigators have endeavored to infect the normal eye with material from the infected eye, but their results have almost always been negative. They have also inoculated other individuals with the same material, but again with negative results. They have then inoculated the normal eye of the monocular trachomatous case with material from binocular cases of trachoma in various stages. With material taken from fresh cases they invariably produced the disease in the normal eye as well as in normal control cases. However, material taken from

chronic cases generally did not reproduce the disease. All this tends to the conclusion that trachoma is probably communicable for only a short period in the early stages. If so, as Roche[48] points out, trachoma would not differ from other contagious diseases. This hypothesis would not only explain monocular trachoma, but also the irregular results of inoculations of both humans and experimental animals. It would also explain how one member of a family might have trachoma for years while other members of the family remained free.

It has also been observed by several ophthalmologists that many cases of apparently monocular trachoma are not monocular at all when the "good" eye is examined carefully with the slit lamp. Mossman[49] recently saw pannus as the only sign of trachoma in the "normal" eye of a man with supposedly monocular trachoma. Such evidence strongly supports the theory of a delayed or late infection of the second eye, with either a diminished virulence of the infective agent, or a partial immunity in the host by the time that eye became inoculated. Heymann and Rohrschneider[41] believe that it is the virulence of the material that is the chief factor in successful inoculations, but they also emphasize the fact that such material must not be taken from later stages of the disease.

According to Krumbach,[50] individual differences in receptivity to infection are apparent in trachoma, as they are in many other infectious diseases. This, of course, would not at all explain monocular trachoma and the absence of the disease in many families of children whose mothers had been seriously affected for years. Regarding this matter of receptivity, it is well to recall the fact that full-blooded negroes are immune or practically immune to trachoma. In the few cases of negroes who have been reported as having trachoma, the possibility of their having mixed blood would be the easiest explanation.

Consistent with the fact of the well-known immunity of the negro to trachoma I have on a number of occasions during the past few years referred to a possible relationship between the amount of pigment in the skin of individuals and races on the one hand, and relatively immunity to trachoma or the degree of infection on the other. Generally speaking, my observation has been that the lighter complexioned the individual the severer the symptoms, and the darker the individual the milder the

symptoms. Many exceptions may possibly be cited to discredit this hypothesis, but if we consider the effects of trachoma among large groups, perhaps among races, I believe that the principle suggested will hold good. For example, the severest form of trachoma that I have ever seen is that affecting the mountain white in the Ozarks of Missouri. These people are mostly the fair-haired descendent of early American settlers of Nordic extraction, chiefly English and Scotch. Rice and Smith[51] of the U. S. Public Health Service estimate that 3.9 per cent of the 17,000 active and inactive cases of trachoma in the state of Missouri are blind, under the present legislative definition of blindness, which is limited to those not having vision better than an ability to see hand motions at one foot — admittedly a very severe standard. They found 12.5 per cent with vision as low as 20/200 (industrial blindness) or worse.

Let us now take an example of a darker-skinned race. In 1927, I[52] made an estimate, based upon observations in different parts of China and data secured from certain records, books, and governmental officials, that there were approximately two million blind Chinese, about half of whom were made blind by trachoma. I further estimated that there were probably one hundred million Chinese with trachoma, calculated upon an incidence of twenty-five per cent of the population. On this basis, therefore, about one per cent of those afflicted with trachoma in China become blind. I should add that approximately the same definition of blindness was employed as that used in Missouri. A comparison of the figures shows that about four times as much blindness results from trachoma among the white of the Ozarks as among the Chinese. Judging from my own observations in Japan and from published reports the disease in that country seems to be more severe than it is in China, but not as virulent as it is in the Ozarks. It is also more severe in India and Egypt than it is in China, and much worse in southern Europe. Its devastating effects are approximately the same among the American Indians as among the Chinese.

Based, therefore, largely upon my own observations, which I must admit are in no sense complete, I would place the races in the following order according to the severity of the symptoms and the sequelæ of trachoma: 1. American whites as typified by the people living in the mountains of Missouri, Kentucky, and

Tennessee; 2, Peoples of southern Europe; 3, Japanese, Egyptians, and natives of India; 4, Chinese and American Indians, 5, Mixed-blooded negroes; 6, Full-blooded negroes — supposedly immune.

If this hypothesis be correct, and I should emphasize that it is only an hypothesis, it is possible that the amount of pigment in the skin and the conjunctiva is the important factor in immunity or in susceptibility to trachoma. If so, it does not seem unreasonable to suggest the therapeutic use of pigment inoculations in trachoma. A recent article by Testu[53] implies that he has treated trachoma by using the blood of negroes.

V. Etiology:

The etiology of trachoma still remains obscure despite the continued painstaking researches of numerous investigators of the past two generations. So obscure, in fact, is the causative agent that opinion is still divided as to whether trachoma is an infection, or whether it is a virus disease, or a disease of nutritional deficiency, or even an allergy. While the balance of the evidence strongly indicates that trachoma is communicable and probably arises from a bacterial origin, it must be admitted that this concept is not universally accepted. It may be well, therefore, to summarize the latest work and opinions and state the present position of each etiologic theory at the present time.

A. Inclusion Bodies:

No discussion of trachoma would be complete without reference to the inclusion bodies of Prowazek - Halberstædter.[54] Twenty-five years have elapsed since these authors announced their discovery, and still no conclusive evidence has been advanced to prove or disprove them as the cause of trachoma.

A study of the literature shows that inclusions have never been found in cases of folliculosis, but that positive findings in trachoma range from 50 to practically 100 per cent, varying with the different workers and the types of cases examined. In a series of 164 cases of initial trachoma having acute or subacute symptoms Taborisky[33] found inclusions in 163. In his serial studies, which included cases of initial trachoma, suspicious trachoma, chronic trachoma, both with and without scar tissue formation, he found the PH bodies in 67.3 per cent. In the eye

clinic at the Rockefeller Hospital in Peking, my assistants and I found inclusions in 65 per cent of consecutive cases which included all stages of the disease. Approximately half of the cases showed inclusions on the first examination, the additional 15 per cent of positives being found in material secured on the second or third subsequent visits. We also demonstrated, as did Taborisky and others, that inclusion bodies can be found in almost 100 per cent of cases in the initial stage. This is significant in view of the occasional difficulty in making an early diagnosis from the clinical signs. It is important too that inclusion bodies are found most plentifully in conjunctival areas which most inflamed and in cases in which the process is the most acute.

The significance of epithelial cell inclusions in trachoma is, however, greatly lessened by the finding of morphologically similar bodies in inclusion blennorrhea, swimming-bath conjunctivitis, vernal catarrh, Samoan conjunctivitis (epitheliosis desquamativa) and hog cholera; also in cells from the genital tracts of adults with inclusion blennorrhea or of parents whose newborn children have the affection. Hence, it is impossible to believe that the inclusions are specific for trachoma alone.

Although we lack sufficient proof that the PH bodies have a living independent nature, it has been proved, according to Heymann and Rohrschneider,[41] that they are not mutational forms of gonococci or other hemoglobinophilic organisms as was contended twenty years ago by Williams[55] and others. Commenting also on Lindner's[48] theory that inclusion blennorrhea is a genital trachoma, these authors do not believe that proof is sufficient for considering the virus of inclusion blennorrhea identical with trachomatous virus in spite of certain cross-immunity experiments done by Lindner.

The question of "trachoma bodies" cannot be dismissed on the assumption that they are identical with the epithelial-cell inclusions induced by such irritants as nicotine, croton oil, etc., as recently reported by Gifford and Lazar.[56] In our laboratory James and the writer were able to produce epithelial cell inclusions in rabbits with these irritants mentioned above and with a number of others, but both morphologically and in their staining reactions they differed from trachoma bodies. They never took the deep blue stain of the inclusion bodies in trachoma, but were

usually purplish in color. Furthermore, they appeared to be an extruded or split-off part of the cell nucleus. An examination of Gifford's and Lazar's photomicrographs and colored drawings on page 474 of their article shows the nuclear splitting in cells from their rabbits with artificially induced conjunctivitis, in contrast to the apparently whole nuclei shown on the same page to illustrate the inclusion bodies in inclusion blennorrhea.

Several investigators have demonstrated that certain irritants bring about a rapid increase in the chromatin of the nucleus in consequence of which the cell, in its effort to balance the nucleocytoplasmic tension ratio, is forced to extrude nuclear material into the cytoplasm. Horning and Miller[57] in their studies of cancer cells show all stages of nuclear constrictions up to a complete splitting of the achromatic nucleus into two closely approximate halves. An analogy to the effects of irritants on the conjunctiva might be referred to in the contribution of Walker[58] who, when working upon artefacts as a guide to the chemistry of the cell, attempted to demonstrate that the Golgi bodies and mitochondria were artefacts because he could produce by chemical means cellular structures having a similar morphology. His conclusions have already been entirely refuted.

There are certain authors who suggest that the inclusions of trachoma and other diseases may be morphologically alike but that they may be biologically different. Taborisky[33] thinks that these all belong in a related group like the variations of the dysentery bacillus which are differentiated only by their biological reactions. Animal experimentation has thus far failed to solve this problem. Horning[59] suggests that by the use of the new method of micro-incineration of tissues a specific biological difference might be demonstrated. If one found a difference in the inorganic structures he could assume a difference in the organic constitution. This method could also be applied to determining whether the inclusions produced in epithelial cells by the use of chemicals are biologically related to trachoma inclusions.

A recent statement by Wolchonsky[60] epitomizes the present situation regarding these bodies in a very fair and justifiable manner: "Although the etiologic significance of inclusion bodies for trachoma is still disputed and perhaps doubted by more and more, their diagnostic value still remains almost undoubted."

B. Nutritional and Constitutional Aspects:

It has been suggested by certain ophthalmologists that trachoma is a disease of nutritional insufficiency. This hypothesis has been based almost entirely upon the clinical observation that the races and individuals affected are chiefly those living in relative poverty and squalor, and accustomed to a low standard of living. Experimental work to shed light on the possibility has only recently been undertaken, and it is, therefore, impossible to make a statement with positive assurance.

From experiments conducted in Noguchi's laboratory on the effect of vitamine A deficiency in monkeys, Miss Tilden[61] concluded that there was no relationship between trachoma and lack of this vitamine. Later Kendall and Gifford[62] reported that rats and guinea pigs rendered vitamine A deficient were not susceptible to conjunctival infection with Bacterium granulosis. As far as we know these are the only references on this subject, and in both instances, the experiments are not entirely satisfactory because the animals died too soon either of tuberculosis or from too great a depletion of vitamine A. Further work is necessary before this possibility is exhausted. Experiments bearing on nutritional deficiency as an underlying factor in trachoma are being conducted by Dr. Rossleene Hetler in our laboratory, but her work is still too incomplete to allow of any conclusions.

An attempt has been made to study this subject clinically by maintaining patients on a balanced diet including plenty of cod liver oil. Dr. Rice[63] of the U. S. Public Health Service has been conducting such a study at Rolla, Missouri, and at Richmond, Kentucky. Patients have been kept on a diet for six weeks during which time one eye is treated and the other allowed to go untreated. Dr. Rice's impression from these initial experiments is that no improvement has been shown in the untreated eye.

The observation frequently made regarding the high incidence of trachoma among the professional wrestlers of Europe and America certainly casts serious doubt upon the theory of trachoma being a dietary deficiency disease. For this group of athletes obviously has as substantial a diet as any group in the world.

The connection which Angelucci[64] tries to establish between trachoma and lymphatic diathesis is not confirmed by the experience of MacCallan[65] who declares that "neither defective diet nor a depressed physical condition including the so-called adenoidism, is an essential factor in the process of trachomatous infection. It is usually accepted that any depressed physical condition presents a more favorable culture medium for the infective principle of contagious disease than perfect health. It may be concluded, therefore, that this may be the case with trachomatous infection of the eye and its appendages." Boldt, Cirkovski, et al[66] found that 97.6 per cent of trachoma cases examined by him had normal constitutions and concludes that there is no constitutional difference between the trachomatous and non-trachomatous.

Pascheff[5] takes the opposite point of view along with several others when he states his belief that trachoma is not an exogenous parasitic disease, but rather an endogenous constitutional disease characterized by lymphatic-adenoid vegetations of the conjunctiva and cornea with follicular formation which is not a mere inflammatory lymphatic infiltration.

Cattaneo[67] examined very carefully over two hundred trachomatous children and about the same number who were trachoma-free, all of whom lived in the same neighborhood and had an equal opportunity to become infected. He found that although both groups showed a lymph-adenoid constitution, the trachomatous showed a somewhat higher degree of involvement. He concludes that lymphatism and adenoidism mark the whole constitution with their imprints and increase the susceptibility to trachomatous infection.

Those who consider a lymph-adenoid diathesis as the basis for certain follicular affections must be somewhat disconcerted by the statements of several investigators to the effect that folliculosis is an infectious process which can be transmitted not only to humans but to animals. Axenfeld[68] reports having infected himself with folliculosis and Nicólle[69] concludes from the results of his own experiments that the affection is transmissible. Lindner inoculated himself with an organism, alleged to be Bacterium granulosis, obtained from a case of folliculosis by Rieger.[70] The inoculation produced a mild folliculosis which persisted for a year before it disappeared.

Several Italian workers take other points of view from those expressed above. For example, Calendoli[71] cites cases in an attempt to show that many trachomatous affections point to manifest or hidden endocrine disturbances. Bardanzellu and Trovati[72] have come to the conclusion that the trachomatous nodules and papillæ of the conjunctiva are closely related to, if not identical with, the toxico-infective tuberculous granulomas and may be explained on the same basis. I cannot reconcile such a conclusion with the fact that the negro, who is exceedingly susceptible to tuberculosis, is immune or practically so to trachoma.

C. Bacteriological Aspects:

The original bacteriological researches in trachoma were made by Robert Koch after the eminent successes he had achieved in the study of other diseases. He discovered almost simultaneously with Weeks what is now commonly known as the Koch-Weeks bacillus, but which appears to be present only accidentally in trachoma. Since Koch's time numerous bacteria have been advanced as the cause of trachoma by different workers, but in no instance has the evidence offered in support of these organisms been found conclusive or convincing. More recently, Noguchi[42] discovered in American Indians suffering from trachoma a new species which he called Bacterium granulosis and attributed to it a casual relationship to the disease. His reasons for accepting Bacterium granulosis were (1) the frequency of isolation of this bacterium in trachomatous cases, and (2) the appearance of follicular conjunctivitis in monkeys following intraconjunctival injections of this organism. While ophthalmologists examining Noguchi's monkeys were not in accord as to whether the conjunctival changes could be regarded as trachoma, nevertheless, Bacterium granulosis immediately became the most tangible and promising of the bacteria isolated as the possible cause of trachoma. It must be remembered that up to the present time this organism is the only bacterium shown capable of inducing a follicular change in the conjunctiva of the monkey and chimpanzee. Prior to Noguchi's experiments, the only transfers to laboratory animals were accomplished by inoculation of whole material or, as Nicólle[69] reported, by filtered material. And even in these instances there is no evidence that corneal involvement occurred in the animals used for injection.

Noguchi's work has been taken over by Olitsky[73] and his associates at the Rockefeller Institute, and their results not only support Noguchi's original observations but show in addition that secondary infection superimposed upon experimental infection by Bacterium granulosis induces a severer form of granular conjunctivitis which more closely simulates human trachoma. They have found also that intracorneal injections of Bacterium granulosis does stimulate the formation of a "nebulo-vascularization of the cornea," but it must be stated that the illustrations they present do not show the vascularization to be typical of the pannus occurring in human trachoma.

Noguchi's discovery stimulated renewed investigations of trachoma. While occasional reports have confirmed the isolation of Bacterium granulosis the balance of the work has disclosed a complete absence of this organism in trachoma. On the other hand Bacterium granulosis is reported to have been isolated by some from folliculosis, and, as already pointed out, is considered by Lindner and Rieger[70] to be a cause of follicular conjunctivitis. Animal inoculations with Bacterium granulosis have in general, but not in their entirety, been disappointing. Attempts have been made by some investigators to induce trachoma in humans by injections of Bacterium granulosis but except for one reference, the results have been flatly negative. The instance of positive transfer was reported by Addario[74] whose work with one volunteer is not clear or convincing.

In our own laboratory, attempts to isolate Bacterium granulosis by Julianelle and Harrison from patients in Missouri, Kentucky, Tennessee, Georgia and Oklahoma have been unsuccessful. Attempts to transmit trachoma to monkeys by injections of Bacterium granulosis have also been quite inconclusive. Moreover, Weiss was unable to induce trachoma by injections of Bacterium granulosis in three human volunteers while studying material in Tunis.

The argument, therefore, in favor of Bacterium granulosis does not seem very strong when we remind ourselves of just two things: first, that trachoma is rather readily transmitted among humans in whom it often begins with acute symptoms and almost without exception ends in scar tissue formation and corneal changes, but is very difficult if at all possible to transmit to monkeys; second, that Bacterium granulosis occasionally produces

a granular conjunctivitis and in rare instances cicatricial tissue in monkeys, but does not produce in humans even a granular conjunctivitis to say nothing about scar tissue and pannus, which are the characteristic signs of human trachoma.

If we may summarize, therefore, the result of the bacteriological investigations of trachoma, it may be said that while a number of bacteria have been offered by different investigators as the cause of trachoma, sufficiently conclusive proof has not been submitted in support of these organisms. The possibility of Bacterium granulosis as the etiological agent must still hang in the balance until further studies have been pursued. The experiments undertaken are complicated by the lack of corneal changes following intraconjunctival injections of Bacterium granulosis. Whether this fact is an inherent incapability of the organism, or whether the experimental animal is unsuited for the transfer of trachoma still remains unknown.

D. Filtrability:

That trachoma may be due to a filtrable agent was first reported from Nicólle's[69] laboratory. The experiments undertaken there revealed that the bacteriological filtrates of human trachomatous material contained an agent capable of transmitting trachoma. Since the publication of those experiments other workers have pursued similar experiments with conflicting results, so that it is impossible to say whether or not the causative agent of trachoma is a filtrable virus. It must be remembered in this connection that Olitsky[75] and his co-workers have shown that Bacterium granulosis is filtrable under certain conditions and that follicular monkey tissue as a result of injections of Bacterium granulosis may yield infective filtrates. More recent work on the filtrability of bacteria under different conditions show that bacteria may permeate bacterial filters, thus rendering filtrability an insufficient proof of the presence of a virus in filtrates. The properties attributed by Nicólle to the filtrable agent he describes makes his virus an unusual one.

More recently it has been suggested by several investigators that trachoma is an example of associated infection. This concept holds that a filtrable virus initiates the disease, and subsequently secondary organisms prolong and complicate the disease. While this is an entertaining hypothesis there is no experimental

evidence to support it. It is possible that an early acute stage following a short incubation period as sometimes described in trachoma may be incited by a filtrable virus and that the more chronic and slowly progressive phase of the disease may be aided and abetted by secondary invading bacteria, but only future investigations can reveal the correctness of this hypothesis.

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DISCUSSION

Dr. Martin Cohen, New York: In conducting examinations of the conjunctiva and cornea for the recognition of early lesions and scars, I generally use a corneal loupe, an electric ophthalmoscope with high magnification, or a combined focal illuminator. Since the chronic or usual type of trachoma is seldom observed clinically in its early stages, and the so-called acute type is generally associated with acute congestion of the conjunctiva, it would appear to be difficult, if not impossible, to arrive at an early diagnosis except under rare circumstances. In the future, however, I shall adopt the slit lamp examination in trachoma.

As to the possibility of the complete cure of trachoma, without residual scars of the cornea or partial atrophy of the conjunctiva or tarsus, I have never observed such end results. Fuchs has stated that the final differential diagnosis between follicular conjunctivitis and trachoma in border-line cases depends upon the presence of cicatrization of both conjunctiva and cornea or of the conjunctiva alone. Why this should be so and why it fails to correspond with other diseased conditions elsewhere in the body, I am unable to suggest. However, if complete recovery from trachoma, without cicatrization, is possible, then our conception of this disease as described in standard textbooks should be altered. Cicatrization usually leads to recurrence of inflammation and other sequelæ, though these may be less marked if properly treated and supervised.

Two cases of trachoma have been under my observation for fifteen years, from their incipient stage. One case finally developed atrophy of the tarsal conjunctiva, with ptosis in one eye, but never pannus. The other case developed the same symptoms in both eyes, also without a complicat-

ing pannus. The first patient was examined with a corneal loupe. Possibly examination with a slit lamp would have revealed pannus, but this I doubt as the cornea always remained clear under examination with the loupe and Jervey reports the same experience. I believe that trachoma is primarily of conjunctival origin, as I have never seen pannus without an accompanying conjunctival involvement.

The clinical differentiation between follicular conjunctivitis and trachoma is now better established and recognized. As Dr. Howard says, they are two separate affections, each with its own clinical entities. It is only in cases of superadded acute infections that confusion exists; when the acute symptoms subside the real clinical picture becomes distinct. The presence of follicles with congestion is all that is necessary to diagnose follicular conjunctivitis, their number, situation, etc., being unimportant.

I collaborated for ten years with Dr. Noguchi in the study of trachoma and other conjunctival infections. When, in May, 1927, Dr. Noguchi presented to the American Medical Association his findings on the Bacterium granulosis, I discussed the paper, and at that time reached the conclusion that he has isolated a micro-organism capable of producing in monkeys and chimpanzees a follicular conjunctivitis similar to that brought about by a direct transmission from man to monkeys. I am still of this opinion in the absence of further evidence to the contrary in regard to conjunctival cicatrization and pannus. The latter lesions I have never observed, although I was permitted by Dr. Flexner and Dr. Olitsky to examine the animals from the time of the first inoculation to the present time. I received the impression that the Bacterium granulosis recovered from human trachoma was another organism from the many in the conjunctival sac, and produced in monkeys a condition similar to human follicular conjunctivitis. Whether the condition as produced in animals is the same as human trachoma has not as yet been proven. I understand from Dr. Olitsky that the Bacterium granulosis has been absent in the six cases of follicular conjunctivitis that he has studied, and was also absent in five patients studied by Biette. Lindner states that the organism he used in producing follicular conjunctivitis in two patients and in himself strongly resembles Noguchi's Bacterium granulosis. Many negative findings have also been mentioned. Therefore, it still remains to be proved that the Bacterium granulosis can cause follicular conjunctivitis.

The inclusion cells of Prowazek and Halberstædter, sometimes called trachoma bodies, were also considered originally to be the cause of trachoma. They were recovered in trachoma, rarely and with difficulty, from conjunctival scrapings and in epithelial cells stained with Giemsa solution. Noguchi and I studied and reported on this problem and continued the study for many years. Seventy-five cases were seen in five years at the Randall's Island Hospital, occurring either alone or in association with epidemics of Koch-Weeks infection, pneumococcus infection, influenza and ophthalmia neonatorum (gonorrhoica and non-gonorrhoica). Two cases finally showed a diffuse and linear cicatrization of practically

the entire palpebral conjunctiva for two years, but no corneal or other involvement; two cases showed a papillary hypertrophy of the entire conjunctiva with ptosis, but no corneal complications. Final observation was impossible. Fifty-four cases resulted in a normal conjunctiva. Four cases of follicular conjunctivitis (Dr. Howard) became infected with inclusion cells.

In a paper I presented to the Academy in 1912, on the clinical course of conjunctival affections associated with so-called trachoma bodies, I reported on twenty-four cases in which the inclusion cells alone were found by Noguchi. The infectious disease was of an acute type, though the inclusion cells were present from two to nine months. In cases of ophthalmia gonorrhoica where inclusion cells were found in connection with gonococci, and in some cases of typical trachoma, it was inferred that the diseases caused by these bodies had probably been ingrafted on the original affections.

Our hypothesis that the inclusion cells are responsible for an independent conjunctival disease cannot be absolutely proved until a means is discovered of growing these bodies in pure culture. There is, however, sufficient evidence that the term trachoma bodies is a misnomer and should be discarded.

I have had no personal experience with the filtrable virus of trachoma and the Bacterium granulosis, or with diet in vitamin A deficiency. However, I believe that diet, as mentioned by Dr. Howard and others, has no direct influence in the causation of trachoma or the presence of Bacterium granulosis.

Olitsky, in 1931, in his article on the filtrability of Bacterium granulosis, stated that up to the present time it has not been possible to pass a pathogenic strain of Bacterium granulosis through tested filters, also that when suspensions of trachomatous human and monkey tissues, which contain fewer organisms than do actual cultures, were used, Bacterium granulosis was never recovered from the cultures.

The question of superadded infections or mixed infections occurring in the conjunctiva as possible etiological factors in trachoma was considered by Lawson in the Royal London Ophthalmological Hospital reports as early as 1897.

Dr. Howard's paper is of great value to the profession. It not only reviews the subject to date, but will undoubtedly stimulate research workers to continue their investigations in trachoma with the aim of obtaining more definite proofs of its etiology.

Dr. S. B. Muncaster, Washington: Dr. John R. Lloyd, my associate, saw a case of a foreign student who had been seen by a prominent New York physician; the young man was using a prescription of tannic acid, soda bicarbonate and camphor water. Dr. Lloyd made a diagnosis of trachoma of mild degree and asked me to see the patient with him. I agreed with this diagnosis, since to me it was a typical case of this disease. I saw hundreds of such cases while studying in Egypt many years ago. We excised several granulations and sent them to Dr. Hunter

at George Washington University and asked him to keep searching until he found true "trachoma granules." Dr. Hunter advised us that it was a very difficult task in this case; I shall read his report:

"Crushed preparations and smears were first made and stained with Geimsa stain, but no typical trachoma bodies could be found, possibly due to the macerations of cells in making the preparation. Sections were made after paraffin embedding and stained with combinations of Geimsa and H. & E. and show a well-defined granuloma with marked thinning of the superficial stratified squamous epithelium, portions of which appear to be denuded and the subepithelial connective tissue is infiltrated with a moderate number of plasma cells, polymorphonuclear leucocytes, many proliferated endothelial cells, together with numerous lymphocytes, many of which contain deep heavy staining nuclei. There is also a moderate proliferation of new-formed scar tissue and some evidence of degeneration of many cells present. The capillaries contained in the tissue exhibit considerable dilatation and engorgement with blood. Scattered through the mass there are many relatively large endothelial leucocytes and with careful search under immersion lens, a few of the clasmatocytic types many be found containing coccoid bodies. While the trachoma bodies are relatively rare, the histological picture presented is, in my opinion, rather typical, and the microscopic picture suggests a trachomatous involvement of a moderate degree of severity."

I do not think it necessary to wait for a pannus of the cornea to diagnose trachoma.

Dr. Sanford R. Gifford, Chicago: Since the subject of inclusions produced by chemical irritants has been mentioned, I would like to mention some further work by Dr. Lazar and myself on this subject which has not been published. Our previous report described the production of inclusions in the epithelial cells of animals following the instillation of nicotine and croton oil. These were considered morphologically indistinguishable from the inclusions found in trachoma, although Professor Lindner did not consider them true inclusions. Although they could be found in a number of animals so treated they were never numerous, only one or two typical ones being found as a rule in a cover-glass preparation. Ozuchi and Mikuchi, in 1930, reported the use of a two per cent infusion of Radix Senega, a drug containing saponin, to produce an increased mucoid secretion in suspected cases of trachoma, inclusions being especially numerous, according to these authors, in such secretions.

We employed this drug in the eyes of animals and ourselves, producing a moderate conjunctival hyperemia and increased secretion. In rabbits treated for several days with the drug, the cells containing inclusions such as were previously described were more frequent than in our former animals. Some typical examples will show the resemblance of these cells to the cells seen in trachoma, the inclusions staining blue with Geimsa, while the nuclei are pink. A few attempts to produce such inclusions on the adult conjunctiva were negative. The infusion was then used in the eyes of twenty-six infants, several days old, which had shown

no previous discharge. The reaction was not severe and subsided within twenty-four to forty-eight hours. Scrapings made during this time, however, showed in three cases perfectly typical inclusions. One preparation showed four in one microscopic field and a number of others in the slide. These were found for four to five days following the last instillation. Scrapings from normal infants never showed such cells. The preparations were shown to a number of ophthalmologists, including Dr. Wilson, of Cairo, and the members of our department of bacteriology. These gentlemen considered the inclusions produced in the babies indistinguishable from those in trachoma. Professors Lindner and Lowenstein believed there were differences in appearance, the exact nature of which was never made clear to us. Professor von Szily believed that work with moist preparations and sections would be necessary to insure that they were identical, and such work is now in progress. Dr. McKee was good enough to look over some of our slides, although I am not sure if he saw the ones from the babies, and perhaps he may express his opinion on them.

A photograph is presented showing preparations from a baby with pneumococcic conjunctivitis. The fact that many more inclusions were found in these infants seems to indicate a special ability of the infant conjunctiva to produce them during any inflammation.

If these findings mean anything, they mean that inclusions are the products of cellular reaction to certain agents, bacterial or chemical, which produce inflammation. Certainly the bodies in our preparations are not parasites, and it seems unlikely that the similar bodies found in trachoma, gonorrhœal ophthalmia, swimming-pool conjunctivitis and non-gonorrhœal conjunctivitis of the new-born are parasites.

Dr. S. Hanford McKee, Montreal: I realize that a chairman should keep his chair, but as I have been asked to say something I shall acquiesce.

I think the example Dr. Howard gave of the contagiousness of trachoma must indeed have been an extreme case. While no one can deny for a moment that trachoma is a contagious disease, I am under the impression that it is not nearly so contagious as we have been taught to believe. I have had a personal experience such as that described by Dr. Howard, and immediately leaving the case I went into another room, had two per cent silver nitrate instilled into my eye, and I have not trachoma. At least, I am not seeing anybody about it.

I have had very little to say about the Bacillus granulosis because I have felt that I might be misunderstood; but I have felt that B. granulosis was one of the large group of saprophytes found in the conjunctiva. I believe I have seen B. granulosis on many occasions, but I always put the slide aside, believing I had done the Gram stain a little too hastily and that the organisms which should have been Gram positive were for that reason Gram negative.

As to the slide that is sent out routinely and the man asked to find trachoma bodies, in the first place eye bacteriology depends a good deal upon who makes the specimen, and in the second place upon who

examines it; but the more important is the one who makes the specimen. It is only the person who is used to doing that kind of work who can make a specimen so that you will get some definite information, positive or negative, from the laboratory.

Trachoma bodies photographed are rather different from trachoma bodies under the microscope. I mean by that, that when you put a series of slides on the screen the trachoma bodies, both the true and the so-called pseudo bodies look absolutely similar. On the other hand, these slides under the microscope will look differently. I really do not know where I am in regard to trachoma bodies. They are one of the most interesting of pathological entities. I have set up conjunctivitis in rabbits, guinea pigs, etc., but I have never seen, to my satisfaction, inclusion bodies from these cases.

Dr. Gifford was good enough to send me some of his slides, but it was very difficult to give a definite answer, and I am not prepared to give an opinion now, but when I look at these slides of Dr. Gifford's I do not believe they are either the real or pseudo-trachoma bodies. They look so in the photograph, but to my mind they do not look so under the microscope.

Within two months at the Children's Hospital I happened to see a little series of eight cases of ophthalmia. They were all babies a few days old. In three cases the smears were filled with trachoma bodies alone; nothing else could be found on the slides after a long examination. In three others there were numerous trachoma bodies and gonococci; in the other two cases there were only gonococci.

Dr. Harvey J. Howard, closing: If there had been time today to have presented to you my paper in full, I believe I would already have cleared up the points and questions brought out in the discussion. One thing which I did not have time to say is that I do not consider trachoma more than mildly contagious. It certainly is not contagious to the same degree that inclusion blennorrhœa is contagious. The example to which I referred was given for the purpose of showing that trachoma is a transmissable disease. Perhaps Professor Fuchs' case was unusual, but there are a number of cases on record which demonstrate that trachoma has been directly transmitted from one human to another.

I have endeavored to show that most authorities consider trachoma infectious only in the early stages or during the period of an acute exacerbation. The chairman has referred to an accident which happened to him some years ago. One explanation of his not developing the disease is that he probably received material into his eye from a stage of trachoma that is not infectious. It must also be conceded that there is a difference in the receptivity of the individual, as I tried to show today when I introduced the subject of differences in the cutaneous and conjunctival pigment.

In reply to the question of inclusion bodies, I find that I am in accord with our chairman. I do not say that they are parasites, but, on the other hand, I am convinced that their presence in specimens must be considered as significant. In my paper I state that Taboricky found

them in 163 of 164 cases of initial trachoma, and that in Peking my assistants and I found them in 65 per cent of all cases, both early and late. These percentages have largely been confirmed by several other workers. Among the many men who consider trachoma bodies important is Wolchonsky, who says that "although the etiologic significance of inclusion bodies for trachoma is still disputed and perhaps doubted by more and more, their diagnostic value still remains almost undoubted."

I am particularly happy that Dr. Cohen is in such close agreement with what I have brought out in my paper and also that he has so clearly stated his position regarding Bacterium granulosis. I want to thank him and the others who have taken part today in the discussion of this interesting subject.

SUPERFICIAL PARENCHYMATOUS PUNCTATE KERATITIS

WILLIAM THORNWALL DAVIS, M.D.

WASHINGTON, D. C.

THIS FORM of keratitis is usually of toxic origin and is characterized by periodicity, symptoms of ocular irritation and conjunctival congestion.

Etiology—In the eleven cases seen, eight have apparently been due to foci of infection in the tonsils and teeth or both. Of the remaining three, one was due to gastrointestinal infection, one to possible tuberculosis and the third disappeared before a study could be made.

Symptoms—The first case of the series after extensive study yielded the discovery of the characteristic lesion. This patient had a chronically red eye over a period of some twenty years with acute exacerbations of the redness accompanied by a feeling of irritation and lacrimation.

In the usual case the subjective symptoms during an exacerbation are those of a moderately severe conjunctivitis. There is a sensation as of a foreign body in the eye and redness of the bulbar conjunctiva. The upper lid may be slightly edematous which causes a moderate narrowing of the palpebral fissure. There is no chemosis.

The ciliary system presents no symptoms as a rule. The ciliary vessels are not congested, or but slightly so, and there are no cells in the aqueous nor deposits upon Descemet's membrane. Occasionally, there may be seen a slight flushing of the circumcorneal ciliary vessels. In the intervals of quiescence all the subjective symptoms disappear and the eye is quiet. The conjunctival redness may remain, however, so that the patient is annoyed by a continuous moderately red eye. This may not always be so; in the intervals between the attacks the eye may be quite normal in appearance.

The appearance of the cornea is characteristic and can be observed only with the biomicroscope. The lesions are too mi-

nute to be seen by other methods of examination. When the cornea is examined with the slit lamp the characteristic lesions consist of fine, white, angular points in the corneal substance immediately beneath but not involving the epithelium. It is important to stress the fact that the epithelium is not involved. These fine points vary in number from four to six to fifteen or twenty and are usually closely grouped in the lower central portion of the cornea below the pupillary area. Very rarely one may see a few of these points in the upper portion of the cornea. Usually only one eye is involved, but both may be.

By using a very narrow slit and with the high magnification one may localize the fine white points in the most superficial layers of the cornea immediately beneath but not involving the corneal epithelium. These points are of alabaster whiteness, not round, but tend to be angular. They cannot be further resolved by higher magnifications into component gray dots but appear to be homogeneous. On disappearing there is no trace left in the cornea, which appears perfectly clear. They take fluorescein stain very brilliantly so that after staining they have a scintillating iridescence which I have not observed in any other corneal condition.

In certain cases three or four of these points may remain in the cornea continuously and in such an individual there will be some redness of the eye with or without irritation. Usually, however, in the intervals all of these points disappear and the eye is quiet. The epithelial surface of the cornea is not disturbed. There is no marring of the perfect brilliancy and smoothness and no staining of the epithelium with fluorescein. In this it differs from other forms of punctate keratitis hitherto described in which the epithelium is involved.

Occasionally one may see in association with the white points small grayish striate areas in the same level of the cornea as the white points. They do not involve the epithelium nor take the stain; these gray areas resemble somewhat the appearance of the cornea after the use of the tonometer. The white points, however, are the characteristic lesion, the gray area being only occasionally seen in association with them.

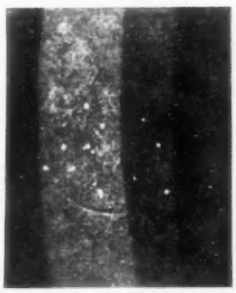
Since I have made these observations I have studied a late edition of Vogt's "Atlas of Slit Lamp Microscopy of the Living Eye" and find he has described in one case both the white points

and the grayish lines which he designates as glass lines. He does not describe this as a clinical entity, but mentions the one case.

The eleven cases cited in this paper indicate a form of punctate keratitis in the parenchymatous substance which is quite definitely due to a toxemia.

There is no description of any condition resembling this so far as I am aware other than Vogt's. There are many cases of punctate keratitis reported but they are all of the epithelial or sub-epithelial type. The differential diagnosis between the type described and the well-known punctate keratitis is as follows.

In the latter the spots are larger and round or stellate, gray, and elevate the epithelium so that with the naked eye and oblique illumination one may discern them. The form described in this paper does not involve the epithelium and cannot be seen without the slit lamp and microscope. In searching for the deeper angular white flecks of this form one should use a magnification of twenty-eight and more than one sitting may be necessary to find the flecks during the quiet interval. In the acute stage they are less difficult to find as they are more numerous and are accompanied by the glass lines, as indicated by Vogt.



Lehrbruch und Atlas der Spalthlampenmikroskopie Des Lebenden Auges, by Alfred Vogt. Plates 604 and 605.

The symptoms of an acute exacerbation may resemble those of relapsing keratitis bullosa in which the patient is awakened in the night or early morning with sharp neuralgic pain, feeling of a foreign body in the eye, severe lacrimation and flushing of the eye. This so exactly describes certain attacks of deep punctate keratitis that one can but wonder if some of the cases of relaps-

ing keratitis are not this form of punctate keratitis; the lesions of the latter being so minute as to be very easily overlooked.

The left eye was involved in nine out of eleven cases. In the remaining two both eyes were involved. One case presented as the chief symptom moderate edema of the left lid. The patient and her friends noted that one eye was smaller than the other. One case had severe blepharospasm for a year and had been treated with irritating collyria which had probably aggravated and prolonged the keratitis. She had to hold her lids open with her hands in order to see.

The local treatment consisted of holocain, mydriatics or cycloplegics, depending upon the age of the individual, rest and the avoidance of light. The thermophore at a temperature of 110° F. for 60 seconds was used with advantage in several cases.

The cause was diligently sought and promptly removed; in nearly all the cases this proved efficacious.

DISCUSSION

Dr. Walter E. Camp, Minneapolis: Dr. Davis is to be congratulated on his description of these cases of this rather rare disease of the cornea. For the benefit of comparison I should like to show some microphotographs illustrating the pathology of the usual type of superficial punctate keratitis which has been so clearly described by Verhoeff.

No. 1 shows a small punctate spot lying immediately underneath Bowman's membrane and thinning the membrane slightly, either by pressure or by lysis. The overlying epithelium is practically normal. The individual cells are slightly separated which may be due to œdema or to artefact. The punctate area itself is composed of a collection of polymorphonuclear leukocytes which are necrotic, due to their being broken up or destroyed by toxins. The groundwork of the entire spot has a bluish granular appearance due to necrosis and liberation of chromatin granules from the cells. The fibres of the corneal stroma are separated but not destroyed. The corneal corpuscles are not proliferating or affected in any way.

No. 2 is through one of the larger spots and shows almost complete erosion of Bowman's membrane. The epithelium over the spot is probably lost by artefact, or by sponging of the eye during operation. There is no attempt at epithelial repair as one would expect if it were lost prior to removal of the eye. The corneal stroma contains a few more wandering cells than normally present. The endothelium beneath the spot shows a fairly even infiltration with polymorphonuclear leukocytes which are in between the endothelial cells and between them and Descemet's membrane. There is no hypopyon.

No. 3 shows a higher magnification of the preceding area, showing the necrotic leukocytes.

No. 4 is a section through the stroma of the iris showing the proliferation of the small arterioles and capillaries of the iris. These are small, newly-formed endothelial tubes many of which are filled with red-blood cells.

On the basis of these changes found in the iris, and also on the character of the corneal spots and their location around the corneal nerves where they pierce Bowman's membrane to reach the epithelium, Verhoeff concluded that this type of superficial punctate keratitis was due to the action of a toxin on the ganglion cells of the ciliary ganglion. The location of these spots near the center of the cornea rather than near the limbus, would substantiate this view. The hyperasthesia of the cornea, the lowered intraocular tension, and the staining of these subepithelial spots with fluorescein, all would tend to classify this disease with the group of neuropathic or trophic disturbances of the cornea.

The clinical histories of the cases described by Dr. Davis are very similar to those of the ordinary superficial punctate keratitis. There is in both a history of focal infection or acute respiratory infection. In both the disease may be bilateral. In both there is chronicity and a tendency to acute exacerbation. In the objective examination, however, the spots differ in size and apparent location, although in the ordinary superficial punctate keratitis many of the spots are extremely small and do not cause any disturbance of the epithelium. I think there can be little doubt that the lesions described by Dr. Davis are due to toxins reaching the terminals of the corneal nerves. It is possible that his type of punctate keratitis is an attenuated form due to a less potent toxin acting over a longer period of time. Metabolic or endogenous affections of an avascular structure like the cornea are rare and I believe must be transmitted along the corneal nerves or their peripheral ganglion cells.

Dr. Oscar Wilkinson, Washington: I would like to ask Dr. Davis how he explains the ability to make a stain with fluorescein when there is no involvement of the corneal epithelium. It seems to me if there is no involvement of the epithelium one would not get a stain.

There is always an objection to the introduction of new terms in the treatment of ophthalmia. I feel, as Dr. Camp has said, that Dr. Davis has some very interesting cases, very likely superficial forms of punctate keratitis.

Dr. W. Thornwall Davis, closing: I intended to state that there was no change in Descemet's membrane, no cells in the aqueous. My presumption is that the reason these fine spots take the fluorescein stain is that the stain may to some extent be absorbed through the cornea, enough to effect a pathological change of this type. That is the only explanation I can offer. Most careful study reveals no change in the epithelium.

As to Dr. Wilkinson's criticism regarding the introduction of new terms, I quite agree. However, I believe that the slit lamp has opened a large field for new terms, and while I am not in position to state that this is a new form of keratitis, it certainly has certain aspects of an as yet unrecognized clinical entity. In order to distinguish this form of punctate keratitis from the ordinary forms already described, one must designate it by name; hence the new term, "superficial punctate parenchymatous keratitis." If Dr. Wilkinson could suggest some other way of designating this as yet undescribed and unnamed keratitis, so that it may be distinguished, I would be grateful.

OCULAR LIPID HISTIOCYTOSIS AND ALLIED STORAGE PHENOMENA

PARKER HEATH, M.D.

DETROIT, MICH.

IT IS POSSIBLE to classify more accurately a considerable variety of ophthalmic lesions through more, though incomplete knowledge of the humoral and cellular elements involved. By correlating recent discoveries in biochemistry, clinical medicine, and pathology, the ophthalmologist is able to simplify his understanding of a variety of disease processes in the eye. From the study of a series of cellular interactions a confusion of histo-pathological patterns arises. However, in these patterns common underlying changes are noted and used as a basis for classifying some ocular disease syndromes. Any position taken must be tentative because of the incompleteness of knowledge. A bibliography and some suggestions along these lines have been offered by the writer before.

By histiocytosis is meant the immune reactions of an intermediate metabolic tissue having properties of phagocytosis prominent in inflammation. These properties are not exclusive with the cells of the histiocytic apparatus.* By storage is meant the reactions following intracellular accumulations, chiefly leading to fibroblastic proliferation and scar. Cellular parts of this process apparently are inter-locked and a consistent series of pathological steps follow.

The mesodermal phagocytosing cell is designated by the following names: macrophage, histiocyte, reticulo-endothelial,

NOTE — Acknowledgment is gratefully made for the use of material from the following: E. Fuchs, L. Pick, R. Rowland, Army Medical Museum, F. H. Verhoeff, T. B. Holloway, Oeller Atlas, A. F. Sladden, B. Samuels, O. Landmann, R. Moeligh, A. R. McKinney.

* The reticulo-endothelial system or histiocytic apparatus is made up of phagocytic cells in the connective tissue and adventitial cells surrounding the blood vessels and in circulation as monocytes; the reticulum of the spleen, lymphatic glands and bone marrow; periadventitial cells of the liver capillaries, lymph sinuses, spleen sinuses, bone marrow, suprarenal, and hypophyseal capillaries.

mononuclear, wandering, adventitial, granule, foam. Single and multiple groups of these large active cells cause destruction by pressure, loss of nutrition, and phagocytosis in the host tissue. Cells relatively fixed structurally in the eye as microglia and Muller's fibers may enter into similar lesions alone or in combination. Mesenchymal cells derived from a vascular exudation, attracted to the scene through foreign body reaction produce more destructive lesions. The histiocytes are not transient cells as are the leukocytes. The initial inflow is defensive and protective, but potentially overwhelming and harmful. The mobilizations and hyperplasias of the histiocytes lead to the formation of non-malignant granulomata. The large motile cells sometimes grouped around blood vessels ingest large amounts of foreign body substance as lipids, pigments, cellular débris, and become fixed or stored. A local nutritional disturbance leads to more cellular disintegration, serum precipitation, and more cell influx. Thus various steps may be continuous in the same lesion. These locked cells are replaced by fibroblastic proliferation and scar. The stored cells may be more destructive than initial activating lesions. Protective and reparative processes thus go awry by an overwhelming response to what is at first a minor disturbance.

There are many kinds of initiating lesions which produce the chain of events noted above — especially in secondary lipid reactions. Some of these factors are congestion and stasis in circulation, hemorrhage, lipid precipitation, angiomatosis, the chemistry of aging process, free pigments, repeated trauma, circulating chemicals such as phosphorus, endocrine disturbances, and chronic infections, particularly tuberculosis and syphilis. By lipid† is meant a class of fatty and fat-like substances insoluble in water, (except soaps) and put in solution by such solvents as ether, chloroform and alcohols. These substances give special staining and chemical reactions. Lipids are necessarily widely distributed and are essential to the life of cell and tissue. They exist as a fine emulsion in cell and blood serum. The coarsening of this emulsion leads to a foreign body reaction, and this apparently is one of the main causes for the mobilization of

† Chemically lipids are divided into (1) Phosphatides: containing nitrogen and phosphorus (such as lecithim, kephalin). (2) Cerebrosides: containing nitrogen, free from phosphorus (as Kerasin). (3) Sterols: nitrogen and phosphorus free (as Cholesterol) or combined with fatty acids as esters; included are neutral fat, fatty acids (oleic, palmic) and soap.

large numbers of mesenchymal cells. Any tissue may show lipidosis and storage phenomena. Variations in the lesions may be brought about by different proportions of the various lipids and kinds of pigments ingested. The time element in these processes is very important in causing differences in appearance, and has led to much confusion in connecting early and late stages.

Older reactions show fibroblastic proliferation, increase in small round and plasma cells with only a few foam cells. There may be seen on section secondary necrosis in areas, hemorrhage, plasma cells, lymphocytes, eosinophiles, free blood pigments, multi nuclear and foreign body giant cells, cholesterin crystal formation, and cystic spaces.

The late stage is a scar or plaque (often pigment fringed in the retina or choroid of the eye) and may rarely show associated calcium deposits. Hyalin scar formation is particularly seen in relation with the glass membranes of the eye. Differences in the lesions are brought about by the different proportions of the various lipids or kinds of blood pigment present, or are determined by the environment. These cellular reactions follow either a primary metabolic disturbance (frequently lipid) or are secondary to local stimuli. The majority in the eye are secondary. Heredity, familial and constitutional backgrounds are important. The eye offers a particularly rich field for the study of disease based upon these mechanisms.

Some Storage Reactions in the Eye

Following are outlined a few ophthalmic storage reactions. In amaurotic family idiocy (Tay-Sach's) the central nervous system is especially affected as a primary diffuse lipoid metabolic disturbance. Particularly affected are the ganglion cells in the retina which are swollen with intracellular accumulations. This acute more diffuse type of foreign body reaction is especially serious because of the nervous tissue involvement, although other tissues are involved. Juvenile and familial macular degenerations apparently show more chronic storage phenomena with similar basic characteristics.

Niemann-Pick's disease has been carefully studied and unquestionably is related to the above, and may even be said to be fundamentally the same disease. Retinal hemorrhage and exudation have been reported as has an association with xanthomatosis.

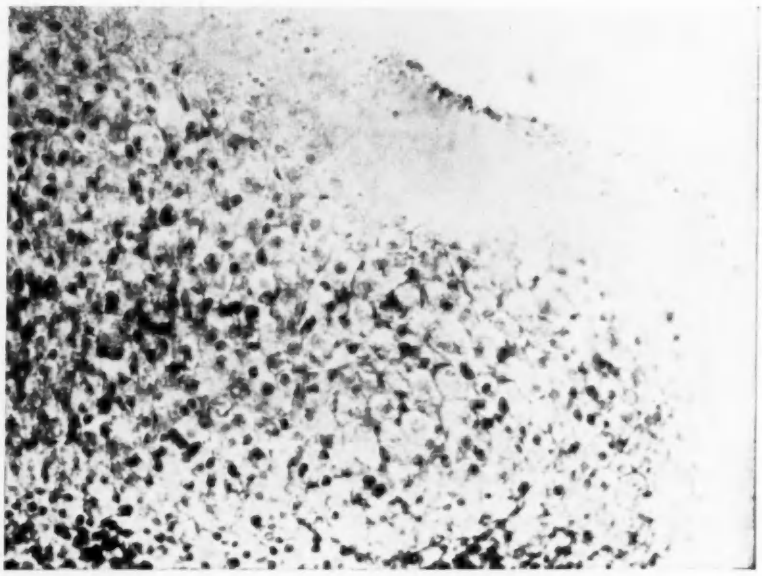


FIG. 1—(Heath)—Amaurotic family idiocy. Section through border of fovea. Primary lipidosis of the ganglion cells of the retina. Similar changes were found in the central nervous system. *(From U. S. Army Med. Museum)*

Schuller's disease and Christian's syndrome of diabetes insipidus with exophthalmos and membranous bone lesions, especially interest ophthalmologists because of the orbital lesions. These consist of deposits of xanthomatotic kernels sometimes associated with loss of the bony apex of the orbit. This juvenile primary reticular lipid storage disease is less diffuse and severe than Niemann-Pick's. Occasionally there may be seen in the fundus retinal hemorrhages and exudations. Association with xanthomatosis has also been reported.

Gaucher's splenohepatomegaly often shows deposits of the modified xanthomatosis type in the conjunctiva, cornea, and sclera, and is a rare form of a still less diffused primary lipo-reticular disease.

The skin of the lids shows two main forms of xanthomatosis. A nodular, comparatively acute form associated with disturbances in sugar metabolism. This lesion can be controlled by diet and exercise. The flat form, chamois leather plaque, is chronic and is not necessarily associated with disturbances in

FIG. 2—(Heath)—Lipidosis (Xanthelasma palpebrarum) of skin. Low grade uveitis associated with lipidosis of the cornea (xanthomatosis).

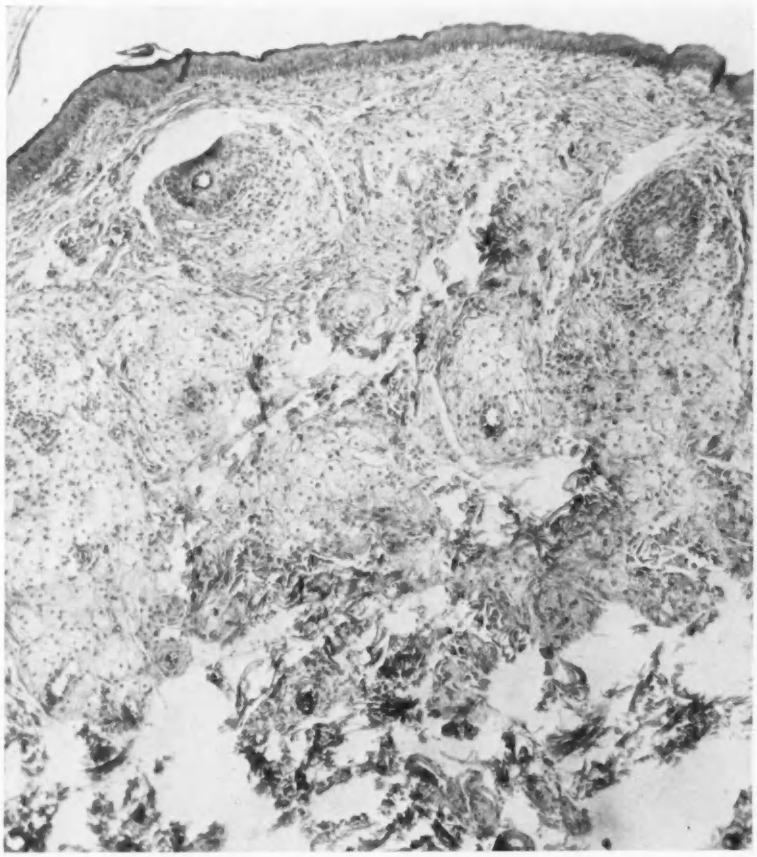


FIG. 3—(Heath)—Section of eye, typical lipidosis (Xanthelasma palpebrarum), many fat-storing cells. Fairly early lesion.

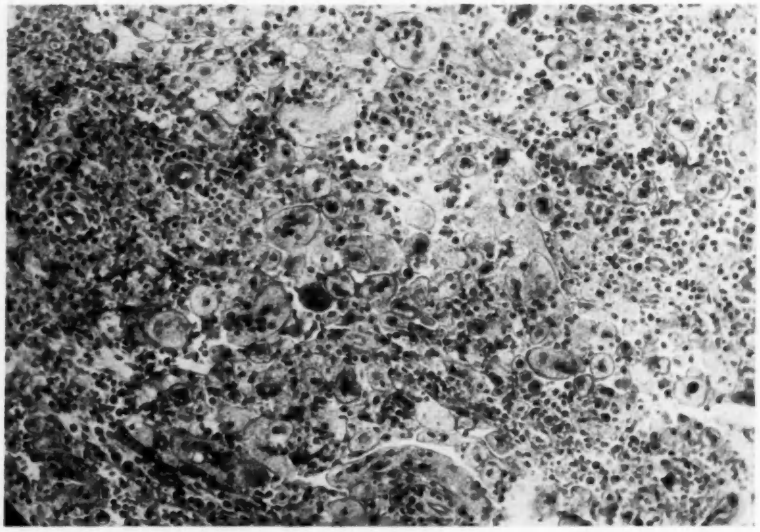


FIG. 4—(Heath)—Higher magnification of later stage of lipidosis of skin. Foam cells, multi nuclear giant cells.

sugar metabolism, and is more purely a storage phenomena of older individuals. It is closely related to the vascular atheromatous plaque. The lid may be thickened from a diffuse lipidosis simulating elephantiasis. Xanthoma are seen in both bulbar and palpebral conjunctiva. On the globe the light yellow tinted waxy plaques are usually found at the limbus.

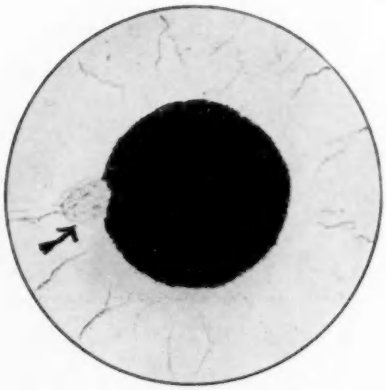


FIG. 5—(Heath)—Xanthoma of the conjunctiva, showing usual location.

FIG. 6—(Heath)—Drawing of section through conjunctival xanthoma, stained with Sudan III. Arrows indicate large cells storing lipids.

The cornea and fundus of the human eye are especially favorable sites to study the origin and development of these protective and reparative lesions. In the cornea, due to its glass membrane, avascular lamellar arrangement, and difference in temperature, more visible variety is introduced in its histopathology. The gerontoxon or arcus senilis is a purely lipid storage process, easily produced experimentally. The lesions of primary lipidosis usually is deep in the cornea. It may consist of transient corneal edema associated with hypercholesterinemia. It apparently may consist of a minor transient loss of transparency associated with an infiltrate of cells. Such a lesion is expansible to a large size. A recurrent type of corneal storage lesion is that seen at the limbus just beyond the vascular loops, usually in patients past middle life. It is a (or multiple) small globular non-staining infiltration, at first fairly sharply bordered lying

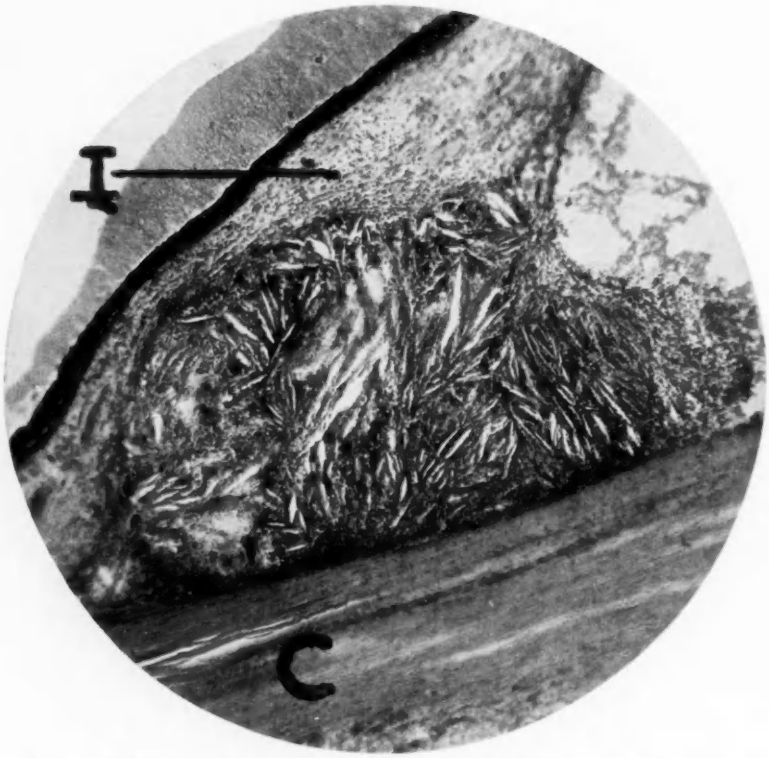


FIG. 7—(Heath)—Lipidosis bulbi, orange-yellow mass in the angle of the anterior chamber, a lipo-fibroblastic mass. *I*, Iris. *C*, Cornea.

anterior to Descemet's membrane. With progression the lesions unite along the arcus and extend fingers centrally, approaching the surface resulting in a loss of epithelium and pain. A superficial and deep vascular ingrowth frequently is added to the lesion at this stage. Regression is slow, partly due to associated uveitis. With some patients it is necessary to cauterize the lesion deeply to prevent dangerous expansion. In lipidosis bulbi the cornea may escape or the major part of it be involved. Usually some areas show yellow or yellow-orange infiltrations, others dense fibroblastic scar, and still others show scintillating intercellular cholesterin crystals. The crystalline elements may dominate in some areas. Fibroblastic lipid masses frequently are part of chronic infections of the cornea as chronic ulcers or old deep scars. The cellular influx in the cornea and anterior cham-

ber from some kinds of a long-retained foreign body, particularly if a splinter of wood, is unusually severe. Clumps of reticular cells are deposited on the endothelium of the cornea in some attacks of uveitis. The anterior chamber angle may be largely filled with fat-staining reticular cells. In some types of long-standing chronic uveitis, cholesterin crystals may be aspirated from the anterior chamber. Xanthomatosis of the cornea according to biomicroscopic observations is a deep stromal lipochrome and crystalline storage.

The sclera shows similar and less severe reaction to active infiltration and storage of these large cells. Old lesions are especially apt to show crystal formation and foreign body giant cells.

In the iris yellow nodules especially with relation to corneal lesion are seen and not necessarily with associated hypotension.

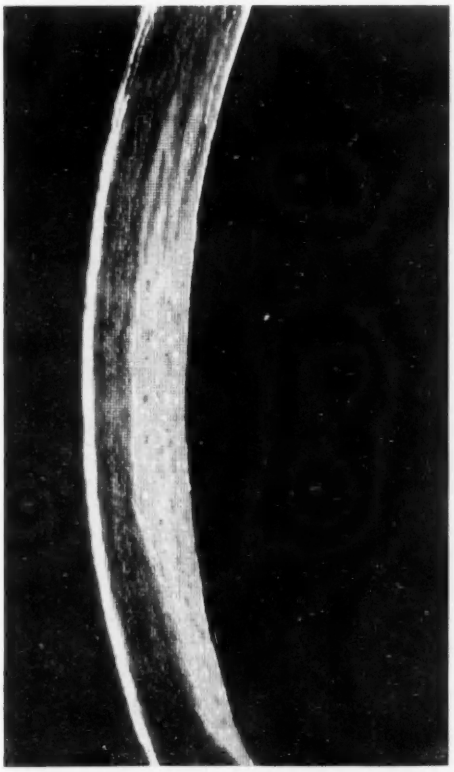


FIG. 8—(Heath)—Xanthomatosis of the cornea. Drawing of optical section. Yellowish deep stromal accumulation, also crystals. Epithelium intact, endothelium pitted.

Animals fed on a high fat diet show large numbers of fat-storing cells in the iris.

The vitreous is the recipient of soap deposits in asteroid hyalitis and numerous cholesterin crystals in synchysis scintillans. In profound accumulation as in lipidosis bulbi, the vitreous contains clumps of enormously swollen reticular cells. These usually show in section a predominence of lipid storage associated with cells storing pigment. The ingestion of pigment seems elective with certain cells. Pinkish yellow exudative deposits behind the retina may detach it and largely fill the vitreous. Among the irregular surface elevations may be seen tortuous small vessels and scattered minute hemorrhages.

The experimental production of drusen in the choroid of animals on a high fat diet has been demonstrated. In the human

FIG. 9—(Heath)—Fat and pigment cell clumps in the vitreous in lipidosis bulbi.

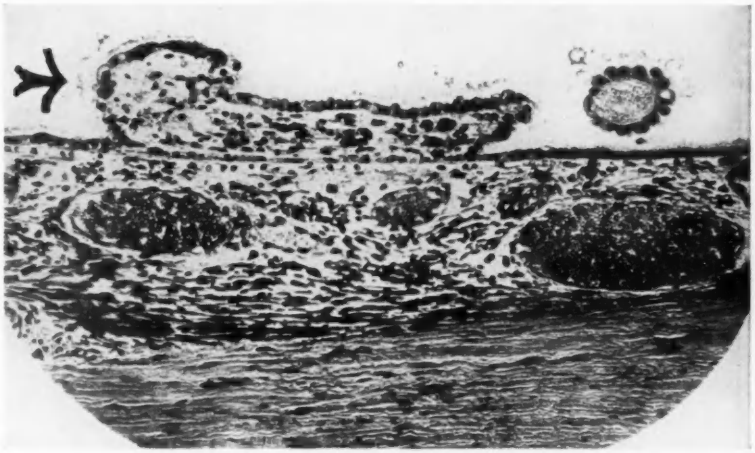


FIG. 10—(Heath)—Drusen in the choroid, cellular stage. Many fat- and pigment-storing cells.

eye drusen are part of the aging process and apparently may be familial and related to the same fundamental changes noted above. Tay's choroiditis is a familial and constitutional form of glass membrane drusen. Honeycomb choroiditis (Doyne's), hereditary hyaline infiltration of the retina (Batten) and tapetoretinal degeneration (Leber) may fall under the broad classification of hereditary lipid storage reaction. Tuberculosis or syphilis in the choroid often shows storage phenomena.

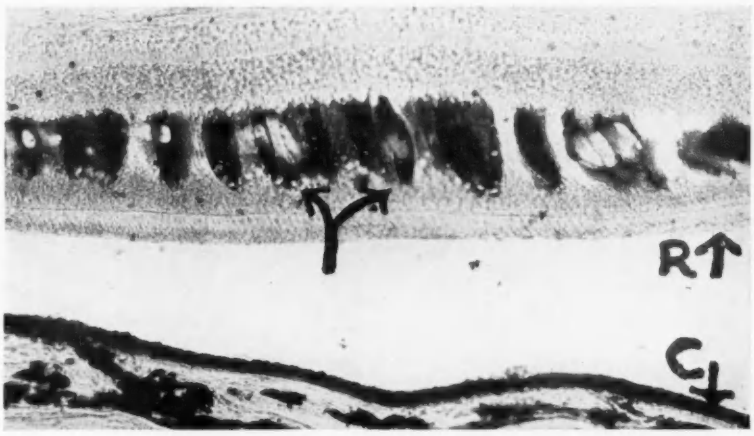


FIG. 11—(Heath)—"Albuminuric retinitis," the fat-selective stain Sudan III emphasizes the large accumulations of lipids. *R*, Retina. *C*, Choroid.

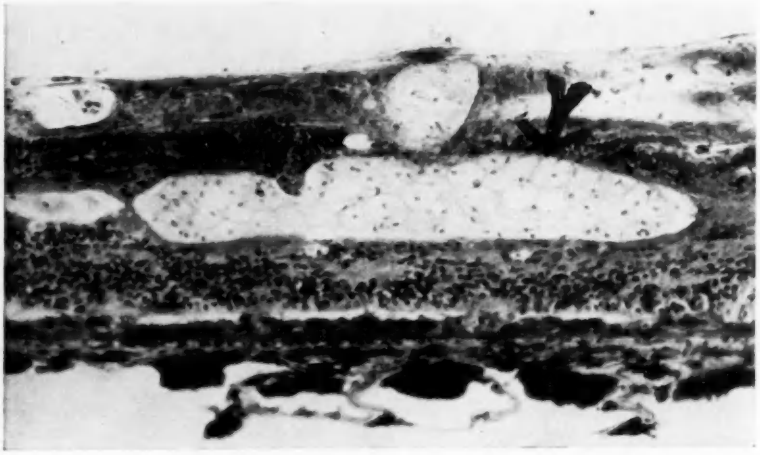


FIG. 12—(Heath)—Retinitis circinata, a vascular cellular exudation in the retina. Large pale cells storing lipids.

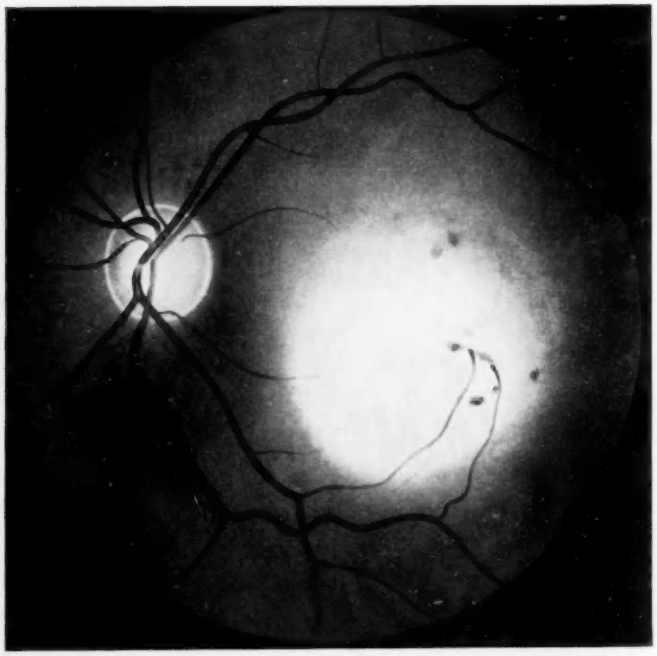


FIG. 13—(Heath)—Disciform degeneration of the macula after Oeller. A fibrous circumscribed plaque in the macula, slow in development.

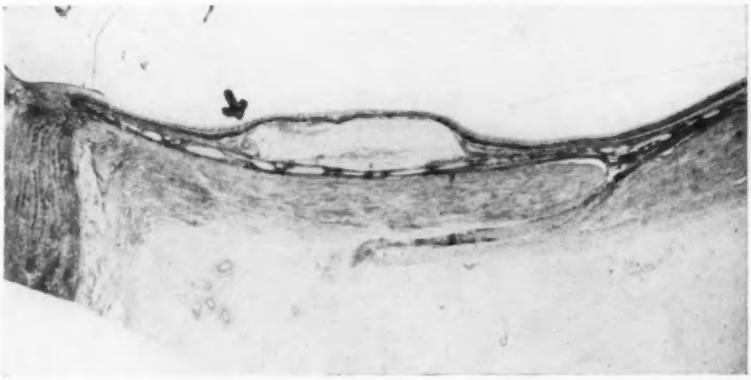


FIG. 14—(Heath)—Section through macular disc. *(From Holloway and Verhoeff).* Apparently the fibroblastic repair is laid down at the same rate that new cells collect at the borders of the plaque.

Arteriosclerosis with exudation has frequently been demonstrated as a fat focus, this is especially true in the retina. Angiosclerosis, circinate retinitis, albuminuric neuroretinitis, histopathologically show the relative importance of foreign body cellular reactions. Sections of disciform degeneration of the macula show fat storing macrophages at the borders of the disc or scar. Fibroblastic proliferation as a gliosis of Muller's fibers has been demonstrated in retinitis pigmentosa, so have the ingestion of free pigment and storage reactions experimentally. Who has not noted that sometimes a minute hemorrhage in the retina seems to cause a relatively large mass of whitish exudate followed by a pigment fringed scar? Exudative storage pathology is thus demonstrated under direct observation. In Lindau's syndrome of retinal angiomatosis associated with cerebellar cyst the retinal exudate leads to a typical storage scar formation. Sections from the liver and kidneys of these familial cases show elaborate active lipid storage in reticular cells. Other forms of exudative choroiditis and retinitis are intimately related. The ordinary microscopical technique frequently shows in section cystic spaces in the retina — previously filled with lipids. Especially retinal cysts are to be found in the ora serrata region of older individuals.

FIG. 15—(Heath)—Section at border of disc showing histiocytic accumulation and fibroblastic repair.

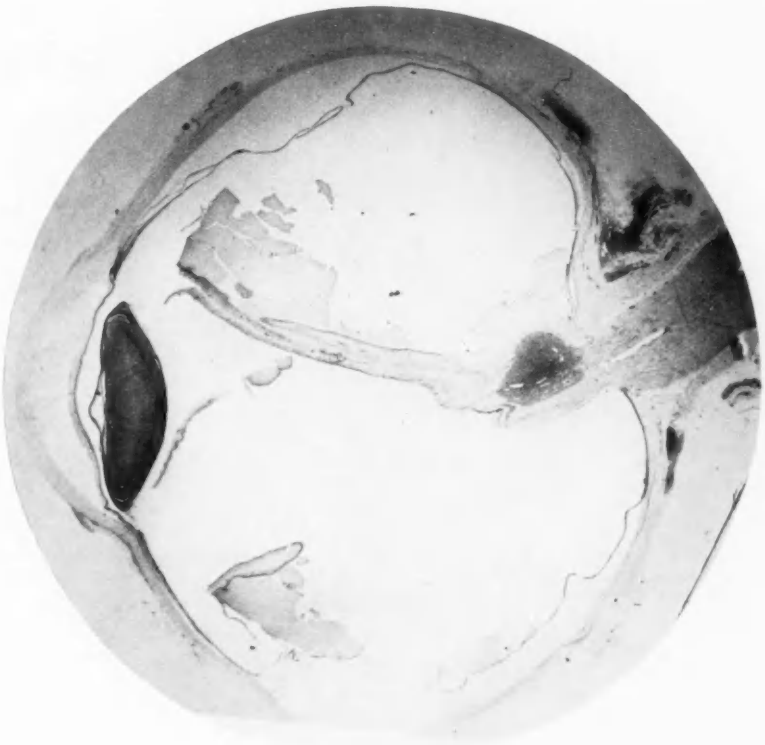


FIG. 16—(Heath)—Lindau's syndrome, retinal and cerebellar angiomatosis. Section through globe in late stage of retinal detachment and scar. *(From A. F. Sladden)*

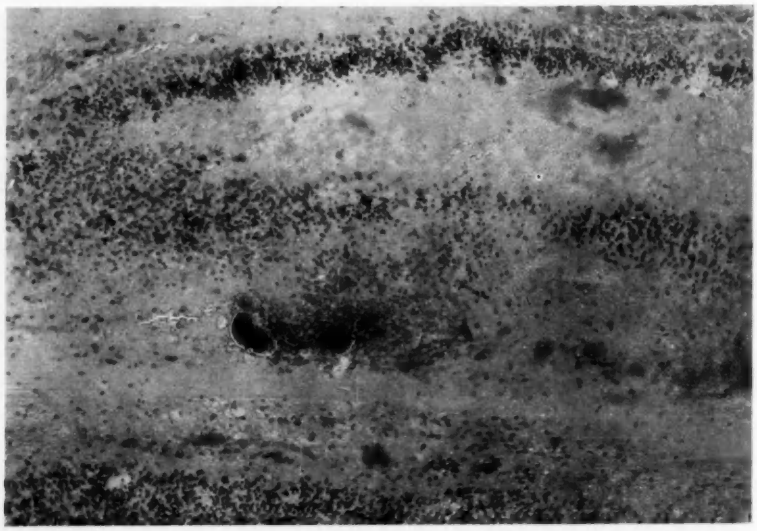


FIG. 17—(Heath)—Retinal scar in Lindau's syndrome. Fibrous with few fat cells.

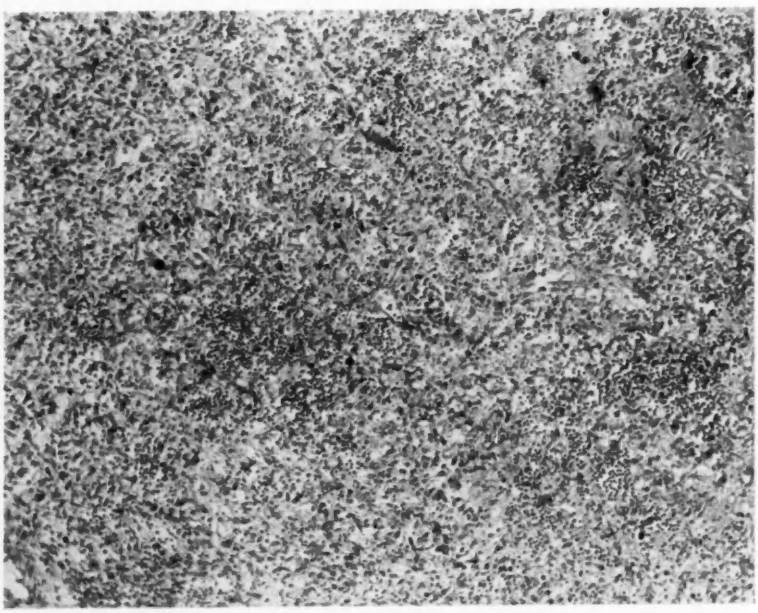


FIG. 18—(Heath)—Left cerebellar lesion. A typical lipidosis associated with a vascular lesion and hemorrhage. A similar cellular influx is found in other organs. *(From A. F. Sladden)*

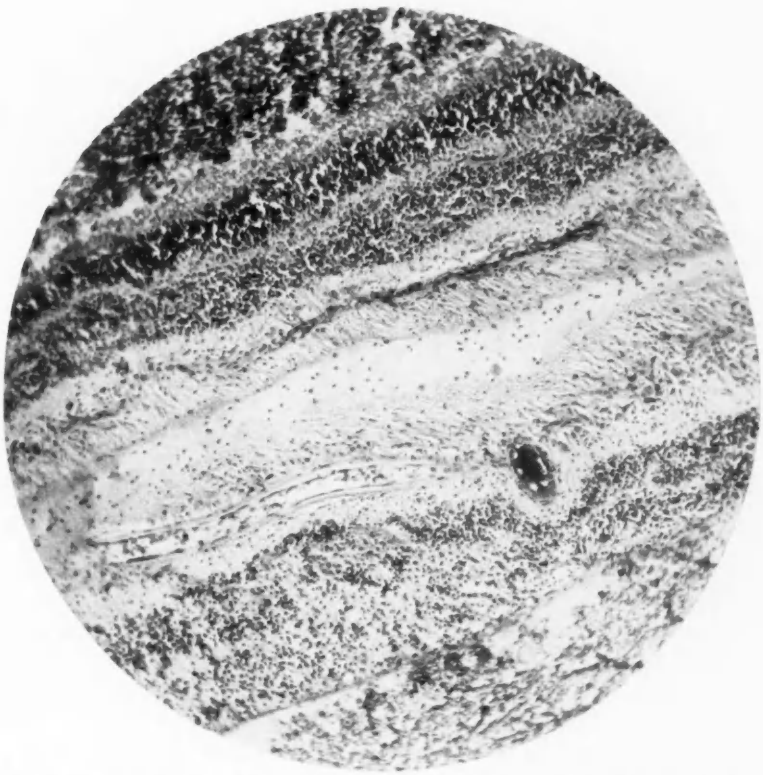


FIG. 19—(Heath)—The retina in Coat's form of exudative retinitis. Extensive lipofibrosis.

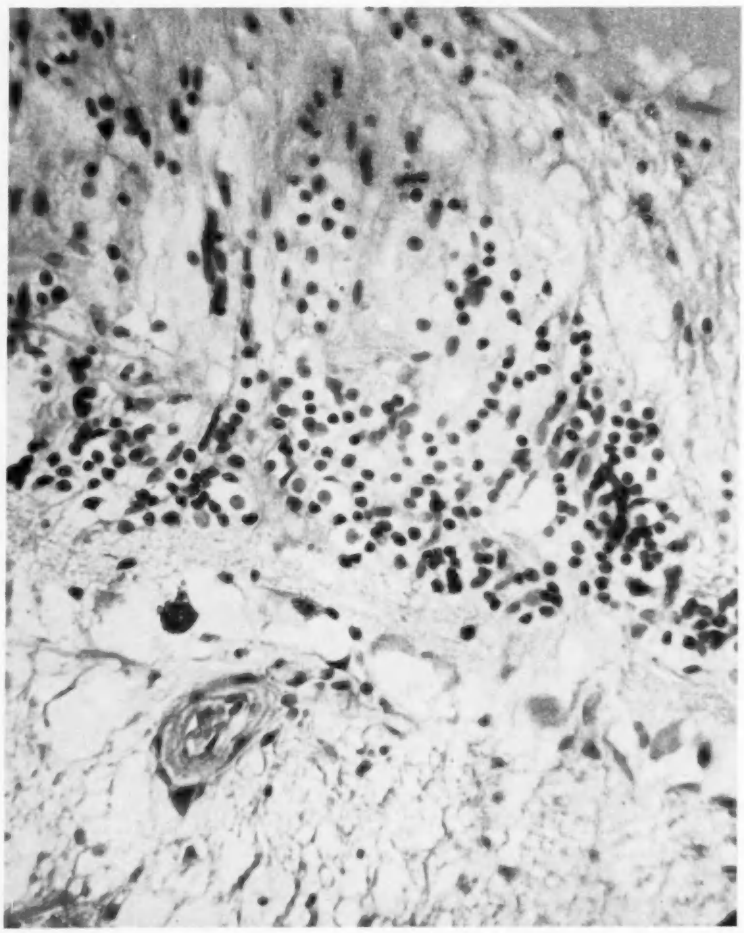


FIG. 20—(Heath)—The retina in Lipidosis bulbi. Fibrosis and fat cell infiltration.

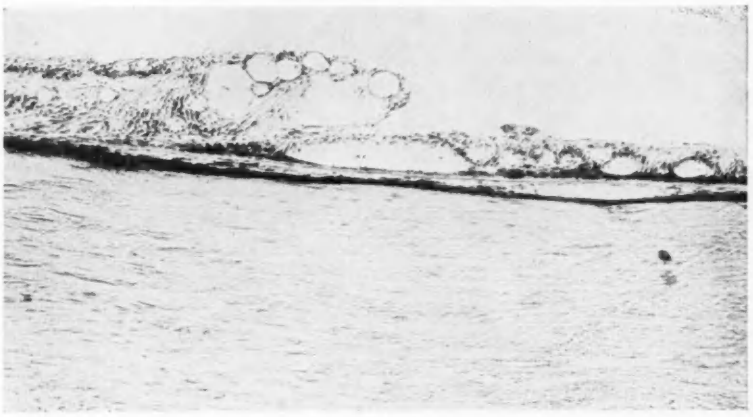


FIG. 21—(Heath)—Cystic degeneration at the ora serrata, part of the aging process. It may be found in sections from other parts of the retina in younger individuals. Fat stains show a high proportion of lipids. In early stages there is an intimate association with large macrophages in groups.

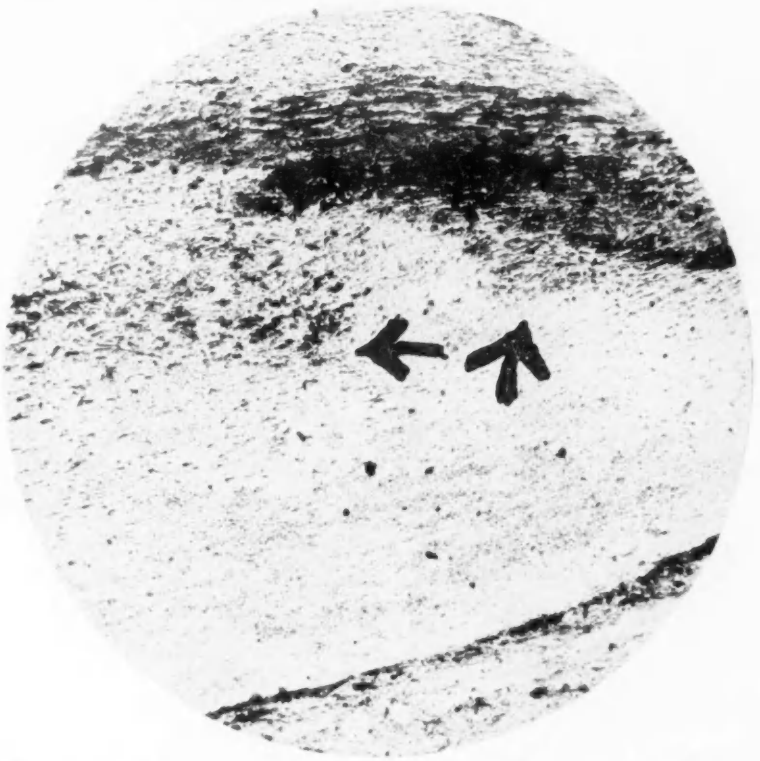


FIG. 22—(Heath)—Sclera, from a case of tubercular uveitis, stained with Sudan III. Arrows indicate proportion of lipid infiltration.

Experimental studies in high fat feeding have been especially valuable in showing some of the mechanisms underlying these cellular activities. The following lesions have been produced: xanthoma, gerontoxon, vascular cellular exudates in the cornea, iris, sclera, choroid, and retina, drusen of the glass membranes of the eye and atheromatous plaques. Tracing the various stages in the storage process has been particularly aided by lipid tinctorial reactions. The importance of fat in these processes is substantial, but is not exclusive. The experimental production

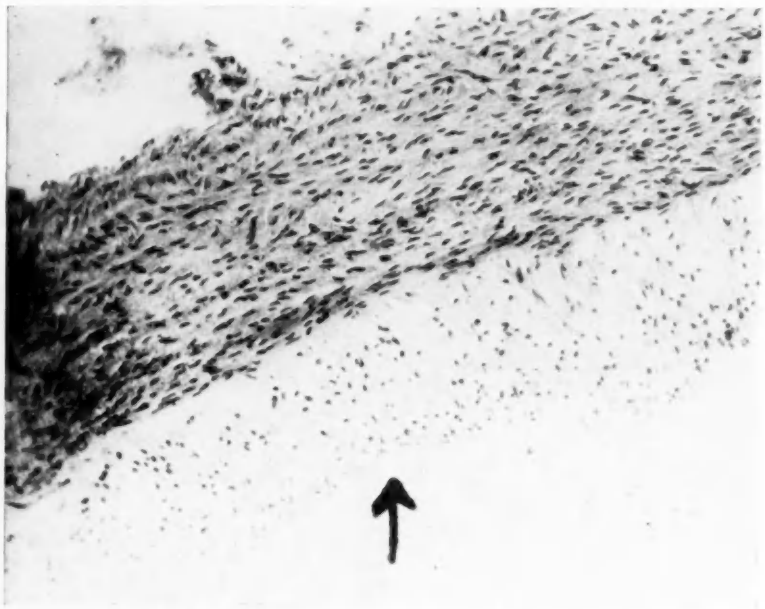


FIG. 23—(Heath)—Aorta of rabbit. Experimental lipidosis from high fat diet. Lipidosis of the intima. *(From Moeligh)*

of pigmentary retinitis has shown the temporary storage possibilities of Muller's fibers and has demonstrated the destructive effects of large groups of macrophages. Other methods have been used, such as the injection of pigments, the use of chemicals (phosphorus) with high fat diet, and gland extracts (pituitary and thyroid) with similar diets.

Summary

An overwhelming immune response of vascular cellular exudative origin often produces a varied but consistent pathological

picture in the eye. The train of events is foreign body substance, cellular exudation, ingestion, storage, fibroblastic proliferation, and scar. The pathological repair is largely brought about by mesenchymal cells. The clinical response varies with the hereditary and constitutional background, age of individual, the kinds and proportions of intracellular substances, and the tissue implicated. Some of the activators of these overwhelming reactions are known. The reaction of reticular tissue in its storage methods varies in different age groups and various disturbed metabolic states. It may be a primary metabolic disease or a sharply localized secondary activity. In the young individual more diffuse activity is noted and in the old a more circumscribed storage phenomenon. Substantial experimental and chemical evidence is accumulating with clinical understanding. The whole process is complicated, and while basic similarities in many isolated ocular syndromes occur we are only beginning to understand cellular and humoral defense reactions.

DISCUSSION

Dr. C. S. O'Brien, Iowa City, Iowa: Dr. Heath has offered us this afternoon further evidence in support of his interesting original observations during the past few years on the pathological changes accompanying disturbances of fat metabolism. I am sorry to have to admit that I have made no systematic study of ocular lipid histiocytosis. It has been only recently, since I was asked to discuss Dr. Heath's paper, that I have taken any particular interest in it. I have had no opportunity to study his slides.

Our conceptions of disease are constantly changing. It has been only a few years since we first heard of the reticulo-endothelium. The histiocytes, or fixed phagocytes, play an important role in tissue response. I am sure we have all noted in our study of pathological processes under the microscope a great similarity in the microscopic appearance of lesions, even though they are produced by widely different causes. One sees evidence of fatty changes in many diseases, but whether they are responsible for further changes, as Dr. Heath believes, or are simply evidence of degeneration, I do not know. It has been my opinion that they were an end process and that they were due to degenerative changes.

Dr. Heath's classification, based on histopathology, may enable us to form certain groups, as he has done, classifying these isolated disease entities into one group, explainable on the same pathologic basis and accounted for by the disturbances in fat metabolism.

I am sorry that I am not better able to discuss this paper. However, I wish to congratulate Dr. Heath on his original and interesting observations.

A STUDY OF THE RELATION OF DISTURBANCES OF CARBOHYDRATE METABOLISM TO CATARACT*

PART I. INTRODUCTION

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NEW YORK, N. Y.

THE ASSOCIATION between disturbances in carbohydrate metabolism and cataract has been demonstrated, but the responsible factors are still undetermined. It was in the hope of contributing facts to this complex problem that the work herewith presented was undertaken. The association of changes in focus of the crystalline lens and the development of cataract in diabetes mellitus are well known. However, all diabetics, even those in the severe stages of the disease, do not suffer from these disturbances.

Duke-Elder[1] differentiated two types of cataract occurring in diabetes mellitus; senile cataract, appearing frequently and at a relatively early age, and diabetic cataract, a rare, characteristic form, peculiar to the disease. The latter form occurs most frequently in the young, when the sugar concentration is high and the patient in a fairly good condition, as a rule. It comes on rapidly and matures in a few days. A few cases of diabetic cataract which cleared after insulin treatment have been reported.

There are many pathological changes in the metabolism of the diabetic organism which may be responsible, in part at least, for the frequently observed changes in the eye. According to Duke-Elder (loc. cit) one of the factors responsible for the high incidence and early occurrence of cataract in diabetes mellitus may be the sensitization of the lens proteins to the denaturing influence of the light. He mentions the increased concentration

* This is the final report by the Academy Research Fellow in Ophthalmology, Dr. Daniel S. Kirby. The work was conducted in the Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, New York.

of dextrose and presence of acetone in the aqueous humor in this connection. Wells[2] also considers that the greater concentration of dextrose in the aqueous humor may be responsible for an increased sensitivity of the lens protein to the coagulating effects of infra-red and ultra-violet rays. In diabetes mellitus the water metabolism of the body is changed which might probably indicate a decreased cellular permeability. It has been shown that one of the effects of insulin is to increase the tissue permeability. Duke-Elder attributes the myopia in diabetes to an excess fluid flowing into and retained in the lens, which swells and is deformed. Actual droplets may be formed under the capsule and impair the transparency of the lens. However, this may be due to a change in the water binding capacity of the lens protein brought about by changes in the dispersion medium such as a slight shift in the pH. In severe diabetic acidosis there is a general dehydration of the tissues to which the decreased ocular tension found in diabetic coma is attributed. The cataract of cholera, in which condition there is a general dehydration, is due to abstraction of water from the lens, according to Fuchs.[3]

The tendency to cataract formation may also be partly due to a deficiency in the auto-oxidation mechanism of the lens. The glutathione content of the blood is slightly decreased in old age and still more so in diabetes mellitus. Glutathione is present normally in the lens and has been reported absent from cataractous lenses.

The carbohydrate metabolism of non-diabetic patients with cataract has been frequently studied. Increased fasting blood sugars and decreased sugar tolerance tests are usually reported in a certain percentage of the cases. O'Brien[4] found a hyperglycæmia present in about one-half of the 218 cases examined. In normal old age there is also a slight elevation of the fasting blood sugar value and a slight decrease in the sugar tolerance.

Disturbances in the fat metabolism are often seen in diabetes mellitus. Ketonæmia and ketonuria are common factors in the more pronounced, uncontrolled cases. The oxidation of the fats and fatty acids is incomplete and the so-called ketone bodies, acetone, aceto-acetic acid and beta-hydroxy-butyric acid circulate in the blood in greater than normal concentrations. The acidosis is increased in the more severe cases and reaches its climax in diabetic coma.

Both sterols and lipides of the blood, of which cholesterol and lecithin are the chief representatives, are often found increased in diabetes mellitus. It has been shown that insulin treatment tends to lower the concentration of these bodies in the blood. Cholesterol and lecithin are also often slightly increased in normal old age.

The greatest increase in cholesterol and lecithin is found in lipoid nephrosis yet there is no undue incidence of cataract formation in this disease. However, one must remember that while in diabetes there is a decreased permeability with a tendency to dehydration and a tendency to acidosis, there is increased permeability and water retention in lipoid nephrosis, and edema is one of the most constant features. The lipide and sterol fractions of the lens as well as the fatty acids have been shown to increase with age. There are many references to increase in cholesterol content of cataractous lenses, up to six and seven per cent of its wet weight. Burdon-Cooper[5] found more cholesterol in diabetic cataract than in other types. Reports on lipide and fatty acid contents of the lens while less numerous also point to a decided increase in cataractous lenses.

The acidosis of diabetes mellitus is due to an excess of ketone bodies. While the pH of the blood is pathologically altered in the more severe cases only, a chronic acidosis may cause a deficit of chloride in the tissues (Peters and van Slyke[6]). The first effect of a sudden increase in blood and tissue dextrose is a retention of water. During sugar tolerance tests one may always observe variations in the corpuscular volume of the blood.

The problem as to the cause of the greater frequency of cataract in diabetes is complex. A great proportion of elderly diabetics develop cataract but in many instances the form and rate of development is not much different from that in the non-diabetic individual of the same age group. The rare condition of typical diabetic cataract is usually observed in the young adult or adolescent. White records only four cases of cataract in her series of diabetic children, three in fatal cases and one in a case of seven years' duration with five attacks of acidosis with coma or near coma. In the diabetic children of the Babies' Hospital no case of cataract has been observed in the past two years, although in one of these cases the blood sugar reached a very high value with marked acidosis and lipæmic retinitis.

Sudden changes in refraction often occur in diabetes mellitus. Duke-Elder summarized the situation by stating that in the course of the disease with high or increasing sugar, myopia tends to occur, while with decreasing sugar after dietary or insulin treatment, hypermetropia tends to occur. It does not happen in every diabetic case and is quite variable even in the same individual. The site of the change in focus is most probably the lens, the capsule and substance of which is freely permeable to dextrose. The fleeting character of the changes does not disprove the theory that dextrose is the responsible factor as it diffuses in and out of the lens quite freely. In this connection one must also take into account the products of dextrose catabolism in the lens and their effect on the water binding capacity of the lens protein. Another hypothesis is that the changes in refraction are due to a change in the index of refraction of the media depending upon the variation in sugar concentration. If the change is regarded as affecting all the media, then the necessary conditions are impossible, but Adler[7] has postulated a differential change in concentration in the aqueous as compared to the vitreous. The sugar concentration of the vitreous is considerably lower than that of the aqueous and since the rate of its changes in concentrations also lags behind that of the aqueous, a relatively small change in either would account for considerable ametropia. Friedenwald[8] could not agree with this hypothesis since the allowable differences in dextrose content of aqueous and vitreous would not account for more than a fraction of a diopter in refraction. Both Friedenwald and Duke-Elder have reasoned that the fluid intake on part of the lens is more effective in changing the refraction when the nucleus of the lens is well developed and while there is still stufficient pliable cortex to respond to the hydropic change. Hence these changes are seen more often in diabetics at ages between forty and sixty years. In connection with these theories one must again consider the effect of an acidotic condition on the water binding capacity of the protein as well as the effect of changes in certain cellular constituents, such as a shift between monovalent and divalent cations, or in the lipide and sterol fractions, on cellular permeability. Severe dietary restrictions have been considered as one of the causes of changes in index of refraction. Jensen[9] reports a case of transitory impairment in refraction due to a strict reducing diet in a non-diabetic woman of sixty-five. In

this case a change in refraction set in three time and each time after an essential change in diet.

Changes in refraction may be accounted for even though the sugar in the aqueous does not rise to any degree. In a number of our patients we found that the aqueous sugar did not follow the blood sugar. In fact, there was often a marked difference. In all the cases examined there was a tendency of the aqueous sugar to approximate that of the blood in the fasting state. In the diabetics the dextrose concentration of the aqueous was often considerably elevated. If the refraction of the lens varied directly with the sugar content of the fluids surrounding it, one would expect a hyperopic change in these patients which, however, is not always present.

Research

Investigations have been conducted by the authors in the study of some of the phases of disturbances of carbohydrate metabolism in patients with cataract are reported in the following divisions.

Analyses of eighty-eight diabetic cases for type of lens opacity and relationship to the severity of the disease or other factors.

Study of the carbohydrate metabolism of fifty-six patients with cataract by means of dextrose tolerance and dextrose depletion (insulin reaction) tests.

Comparison of dextrose concentration in specimen of primary aqueous humor and capillary and venous blood at single examinations.

and

Determinations of changes in the dextrose concentrations of blood, both capillary and venous and aqueous humor during dextrose tolerance and dextrose depletion tests, likewise after food intake. Determination of dextrose concentrations of primary and secondary aqueous humors.

The effect of increased dextrose concentration in the nutrient media of lens epithelium cultures grown in vitro. The effect of lævulose and galactose on such cultures.

The effect of addition of acetone and beta-hydroxy-butyric acid in increasing concentrations on the growth of lens epithelium in vitro.

PART II.

THE INCIDENCE AND TYPE OF OPACITY OF THE CRYSTALLINE LENS OCCURRING IN DIABETIC PATIENTS

DANIEL B. KIRBY, M.D.

THE CASES REPORTED were among those examined in consultation with the department of metabolism. They represent an unselected group of eighty-eight diabetics (22 male and 66 female), whose eyes were examined and an effort made to correlate the findings with the general condition. The age of these patients varied from thirty-one to seventy-seven years. Eight (9%) were in the fourth decade, thirty (34%) were in the fifth decade, twenty - three (26%) were in the sixth decade, twenty - three (26%) were in the seventh decade and five (6%) were in the eighth decade. The known duration of the disease varied from one to sixteen years. They were classified as follows:

	Fasting Blood Sugar mgms. per 100cc.	Daily COH intake gms.	Total daily caloric intake Cal.	Units Insulin per day	Per cent of cases in group
Very mild	80 - 150	115 - 235	1500 or more	none	15
Mild	115 - 160	115 - 235	1500 or more	none	24
Moderate	160 - 180	100 - 150	1500 or more	0 - 25	35
Moderately severe	above 150	less than 100	1500 or more	20 - 60	17
Severe	above 150	less than 50	less than 1500	40 - 70	7

The total carbohydrate intake consists of the grams of carbohydrate in the diet plus 57% of the grams of protein in the diet, converted into carbohydrate by the organism. In judging the severity of the condition account was also taken of the duration of the disease, the mode of onset and the response to dietary restrictions. All patients were under treatment, on special diets or insulin. The pupils were dilated with homatropin 1%. After the examination, a drop of eserin, one-half of one per cent was instilled. The cornea was examined with oblique illumination, the media with transmitted light. Particular observation was made of the lens with the plane mirror at a distance and also

with the + 16.00 or + 20.00 D. lens. The optic nerve disc, retinæ and vessels were then carefully observed. The slit-lamp microscope was not used as it was desired to bring this phase of the work within the scope of the internist.

Summary and Analysis

The Cornea

The corneæ were clear in 66% while there was a slight arcus senilis in 15%, a moderate arcus in 12% and a marked arcus in 7%. The diabetics showed no greater tendency to this formation than the average patients for the age group. Very few had other corneal changes.

The Crystalline Lens and Cataract

The crystalline lens was clear and free from any objective involvement in 36% of the patients. The remainder (64%) showed various types of cataracts. The location of the opacity was in the anterior and posterior subcapsular cortex in 2%; vacuoles or globules in the cortex were found in 47%, punctate opacities in 14%, early water-splitting of the sutures in 23%, separation of the lamellæ in 5%, while the others had progressed to the immature or mature stages, 10% with immature cortical opacities, and 2% with mature senile cortical cataract.

In studying the incidence of lens involvement in relation to the degree of severity of the disease, opacities were found in 40% of the very mild cases, in 66% of the mild cases, in 66% of the moderate cases, in 50% of the moderately severe cases, and in 50% of the severe cases. Lens opacities were found in 70% of the diabetics who showed in addition a recognized condition of general vascular hypertension.

When studied according to the duration of the diabetic disease, lens changes were found in 22% of those in whom the condition was known to have existed for one year, in 62% of those in whom it was known for two years, in 44% of those for three years, in 63% of those for four years, in 40% of those for five years and in 70% of those in whom the condition had been known for more than five years.

The Vitreous

Examination of the vitreous showed it to be clear in 70%. There was a slight haze in 9% due to fine vitreous opacities;

greater haze in 15%; vitreous membranes were found in 6%. There was no case of vitreous hemorrhage. A few patients with fluid vitreous were seen. Opacity of the vitreous was not found to be an important factor in this series of diabetic individuals.

The Blood Vessels

Particular attention was paid to the condition of the retinal blood vessels. To determine the presence of sclerosis the following points were looked for: (1) Indentation of the veins by the arteries at the points of crossing. This feature was present to slight degree in 12% of the cases; moderate in 56% and marked in 14%, a total of 82% of all the cases. (2) Irregularity of the calibre of the column of blood as indicating irregularity of the lumina of the vessels. This was observed in 56%. (3) Constriction or narrowing of the calibre of the arteries observed in 63%. (4) Tortuosity of the vessels in 45%. (5) Increased intensity of the light reflex from the column of blood in the vessels was seen in 41% of the patients. It was found that this latter phenomenon was not a true guide to the presence of sclerosis or thickening and hardening of the blood vessel walls, as the intensity of the reflex was very great in certain mild cases of sclerosis while in certain severe cases the reflex was very dim owing to opacity of the media. The other signs as given in the order above were found to be more constant and reliable in the linking up of the retinal vascular picture with the general vascular condition.

There was general vascular disease with hypertension in 38% of the patients. In these cases, (1) indentation of the veins by the arteries at the points of crossing was present in 90%, to a slight degree in 10%, moderate in 64% and marked in 16%. (2) Irregularity of calibre of the vessels was present in 85%. (3) Constriction or narrowing of the arteries was seen in 73%. (4) Tortuosity of the vessels was present in 51%. (5) Increased light reflex was evident in 49%. (6) Increased fulness of the veins was seen in 19% of the cases.

The importance of the signs of vascular sclerosis followed the same sequence in the cases in which hypertension was part of the condition of general arterio-sclerosis. The signs were present to more marked degree and in a greater percentage of the patients. This emphasized the fact that arterio-sclerosis

both general and local in the eye is an important complication or coincidence in diabetes and that many eye lesions may be ascribed to the vascular disease rather than to the diabetes alone.

The Retina

In the study of all the cases of diabetes, 50% were found without ophthalmoscopic evidence of retinal lesions. Hemorrhages were found in 21%, exudates in 14%, edema in 15%, while changes secondary to previous hemorrhages and exudates were evident in 12%. Healed choroidal lesions were observed in 3%, optic nerve atrophy and detachment of the retina in 1%. The fundus was not visible due to lens opacities in 2%. Retinal lesions were present in 40% of the very mild cases, 45% of the mild, 46% of the moderately severe and in 38% of the severe cases of diabetes under treatment.

Other Complicating or Incidental Disease

Focal infection was noted in 9% of the diabetics while arthritis, rheumatism or neuritis was present in 19%, myocarditis in 14%, syphilis in 8%, and tuberculosis in 1%.

The Coincidence of Lens Changes (Incipient Cataract) and Retinal Lesions in Diabetes

In the diabetics whose lenses were clear, retinal lesions in the form of hemorrhages, exudates, edema or changes secondary to those were found in 40% while in those with incipient cataract, retinal lesions were found in 53%. A greater proportion of diabetics with incipient cataract have retinal lesions than do those with clear lenses, although retinal lesions do not necessarily accompany cataract formation. However, the greater number of diabetics with advanced cataracts probably have also suffered retinal lesions.

The figures on the incidence of arterio-sclerosis in diabetes must make one consider the grave importance of this condition in the production of eye lesions, retinitis and cataract. There are several factors which might explain the great incidence of arterio-sclerosis and hypertension in diabetes. The human ocular capillaries particularly in disease may be less permeable than the normal. This might account for the faulty nutrition of the lens in diabetes.

The Handling of Diabetics Prior to Eye Surgery

The examination of the cases of diabetes for eye lesions demonstrated that the greater proportion of diabetics have arterio-sclerosis. In many it is probable that the retinal lesions and cataract are dependent upon the arterio-sclerosis and renal condition rather than upon the hyperglycæmia. Besiere found that in practically all of his cases of retinitis in diabetes there was also evidence of chronic nephritis. The retinal lesions do not parallel the glycosuria. Insulin treatment controlling the diabetes does not affect the progress of the retinitis. This is practically never seen in a young diabetic. Foster Moore rarely found retinitis before the age of thirty-five years. The longer the duration of diabetes, the greater the degree of arterio-sclerosis.

In nearly all cases of cataract in elderly diabetics, it may, therefore, be said that there is probably also a retinitis. The vascular and renal condition of these patients should be carefully observed and treated rather than simply trying to reduce the blood sugar prior to operation. Many patients will suffer a severe upset and be totally unprepared for surgery if their carbohydrate intake is too markedly reduced or their blood sugar rapidly lowered. The nutrition, energy and reserve of the patients will be better with sufficient carbohydrate and insulin than it will on a low carbohydrate and high fat diet and there will be less likelihood of acidosis.

(The protocols of these cases are omitted from the publication.)

PART III.

REPORT ON CARBOHYDRATE METABOLISM TESTS ON PATIENTS WITH CATARACT

DANIEL B. KIRBY, M.D.

FIFTY-SIX PATIENTS with cataract were selected from the attendance at the eye department of the Vanderbilt Clinic. Thirty had dextrose tolerance tests and sixteen had insulin reaction (dextrose depletion) tests. Ten patients had both tests. The study of the carbohydrate metabolism was made during the day to avoid repeating such work as O'Brien[10] has made on a large series of cataract cases. It was realized that patients of this group may show a normal or average fourteen hour fasting blood sugar and yet suffer abnormally high and postprandial rises so that they are actually in a state of hyperglycæmia from one hour after their breakfast until four or five hours after their evening meal. In such cases there is only a short period from about midnight until breakfast when the blood sugar is within normal limits.

The age of the patients varied from thirty to eighty-four years, three were in the third decade, four in the fourth decade, nine in the fifth decade, twenty-seven in the sixth decade, ten in the seventh decade and three in the eighth decade. The carbohydrate tolerance has repeatedly been shown decreased in advanced age. Thus, Marshall[11] determined the dextrose tolerance in a group of non-diabetic aged individuals. Fourteen per cent of this group gave a typical diabetic curve. The remaining 86% he divided into four groups, (1) normal old age curve 14%, (2) lag curve 25%, (3) storage deficiency (plateau effect in curve) 39%, and flat curve attributable to either decreased absorption from the intestinal tract or increased destruction of dextrose by intestinal flora 7%. He gives the following averages for normal and abnormal old age curves, following the ingestion of 50 gms. dextrose and determining the blood dextrose at thirty minute intervals:

Healthy old age—fasting 103
post dextrose 172, 209, 175, 132, 99
Unhealthy old age—fasting.....109
post dextrose 172, 214, 208, 172, 141

A prolonged elevation of blood dextrose may result in an increased level of the dextrose in the humors of the body as for example the aqueous humor.

Method of Study

The patients were examined at the afternoon clinic, one or two hours after they had had their regular luncheon. Sufficient dextrose (50 grams in about 300 ccm. of water) was orally administered to insure a rise of blood sugar up to or beyond their regular daily post luncheon rise. Capillary blood specimens for examination were taken before and 30, 60, 90 and 120 minutes after the administration of the glucose. In certain diabetics and others, an insulin reaction or glucose depletion test was performed by administering ten or more units of insulin by subcutaneous injection and then testing the blood at the same intervals as given above for the sugar tolerance test.

Certain tests using venous blood were made on patients who visited the clinic in the fasting condition. Again others were given the insulin reaction test in the morning one or two hours after their breakfast.

The Hagedorn-Jensen sugar method with the Somogyi modification for de-proteinization was employed for estimation of blood dextrose.

The results of the tests on the fifty-six cases of patients with cataract allowed their division into four groups. These were no more unusual or irregular than might be found for a similar number of elderly people without cataract.

Results

The group analysis demonstrated that of the total of sixty-six glucose tolerance and insulin reaction tests, 19.4% were in the normal group; 26.9% had the lag type of curve, indicating a slowing up in the removal of glucose from the blood by the tissues; 11.9% had the acicular type of curve indicating a decreased ability on the part of the liver to regulate the entrance of

glucose into the general circulation, while 41.8% had the diabetic type of curve with an abnormally high peak of blood glucose concentration and an abnormally prolonged period for the return to the pre-glucose level. The analysis based on the reactions to the insulin reaction test yielded very similar figures to that based on the glucose tolerance test except that the percentage in the acicular group for the insulin reaction was much less than for the glucose tolerance. In most instances the insulin reaction or sugar depletion can be used as a substitute for the glucose tolerance test and in cases where diabetes is suspected, it will prove the safer procedure. The combined figures bear out the relations. The diabetic type is higher in the insulin reaction group because of the fact that many of these cases were recognized as such and the tolerance test purposely avoided.

The general conclusion is that the carbohydrate metabolism of elderly patients with cataract is not different from that of patients with various conditions in the same age group. A greater number of diabetics are included because of the routine reference of these patients from the Metabolism Department for examination of their eyes.

TABLE

Showng the Results of Carbohydrate Tolerance Tests in Cases of Cataract

Type of Curve	Glucose Tolerance, 39	Insulin Reaction, 27	Total No. of Tests, 66
Normal	8 = 20.5%	5 = 18.5%	13 = 19.7%
Lag	10 = 25.6%	7 = 26.0%	17 = 25.7%
Acicular	7 = 18.0%	1 = 3.7%	8 = 12.1%
Diabetic	14 = 35.9%	14 = 51.8%	28 = 42.5%

(The protocols of the cases on which these figures are estimated are omitted.)

PART IV.

THE RELATION OF CHANGES IN THE DEXTROSE CONCENTRATIONS OF THE BLOOD TO CHANGES OF DEXTROSE CONCENTRATIONS IN THE AQUEOUS HUMOR

DANIEL B. KIRBY, M.D., AND RENEE V. E. WIENER, Ph.D.

CONCEPTS AND THEORIES INVOLVED

An inquiry into the relationship between the concentrations of dextrose in the blood and in the aqueous humor should include:

(1) Definition of the physico-chemical concepts employed.
(2) A resumé of the theories of carbohydrate metabolism.
(3) Discussion of circulation and fluid exchange in the eye.
(4) Discussion of permeability of blood vessels and tissues, and regulatory mechanism.
(5) Examination of the theories of intraocular fluid formation and exchange.
(6) Review of reported analyses of aqueous fluid and blood.

DEFINITIONS

Diffusion relates to the tendency of two liquids or solutions soluble in each other to interdiffuse until they are equally distributed throughout the entire enclosing space.

In the living cell or in the fluid surrounding it the dissolved substances are either present as ions or molecules, or as small molecular aggregates *miscellae*. In either of these states they may diffuse throughout the medium. However, in the living cell this diffusion seems to be regulated and controlled. Not only does the permeating power of the various substances differ, but the dispersion medium may differ in permeability with respect to one and the same substance at different times. The living cell and surrounding fluid represent a many-phased system. Directional permeability, adsorption, metabolic activities, surface

tension and interfacial potential are among the numerous phenomena which influence and determine the rate of diffusion in and around a living cell.

Dialysis is the diffusion of a substance in molecular or ionic dispersion through a membrane impermeable to substances in the colloidal state. In the living organism such membranes are of colloidal nature.

Osmotic Pressure relates to the pressure exerted by a solution placed within a semi-permeable membrane which is permeable to the pure solvent, usually water, only. If such a filled membrane is placed within the pure solvent the solvent will enter until a certain head is established and this is known as osmotic pressure. It is due to the tendency of the solvent to establish the same concentration on both sides of the membrane.

Colloidal Dispersions form a heterogenous system, while substances in true solution form a homogenous system. Many characteristics of the colloidal state are due to this fact. Colloidal dispersions are divided into three classes: (a) Lyophillic dispersions, (b) lyophobic dispersion and (c) an intermediate class. The so called bio-colloids fall mainly into the first group and as the name indicates solvate to a high degree. Solvation, the formation of a compound between dissolved substance and the solvent may be termed hydration if water is the solvent. The majority of the bio-colloids, proteins, gums, starches and lipides have high swelling capacity, lower the surface tension of the dispersion medium, and increase its viscosity. The properties of colloidal dispersions most important to this inquiry are (1) colloid-osmotic pressure also termed oncotic pressure, (2) swelling capacity, and (3) the influence of changes in pH and other electrolytes on these phenomena and on states of aggregation.

In applying the laws of osmotic pressure to colloidal systems one must assume that each colloidal particle carries the same kinetic energy as a molecule. Some colloids such as proteins may be present in molecular state plus the water of hydration. To give a measurable osmotic pressure the colloidal particles must be present in not too dilute a concentration. Lyophilic dispersions exhibit measurable oncotic pressure but the values are very small compared to the osmotic pressure values of true solutions. The only reason why the oncotic pressure of the proteins plays a part in the interchange of water between blood

and tissue fluid is that the osmotic pressure of the blood is compensated by the osmotic pressure of the tissue fluid, while the oncotic pressure of the blood is only partly compensated by the oncotic pressure of the tissue fluids.

Concentrated lyophilic solutions have a tendency to take up more of the dispersion medium and the work which is performed by the colloid in this process conserves energy to a remarkable degree. Electrolytes, especially hydrogen ion, play an important part in this phenomenon, especially in the case of proteins. The physical elasticity of a protein jel also depends upon its concentration and the presence of electrolytes.

Ultrafiltration is the separation of a colloidal dispersion from its medium by passing it through a filter with fine enough pores to retain the particles in colloidal dispersion. Ultrafilters are mostly gelatin or collodion membrane and measurable filtration can only proceed under a high gradient, either high pressure or vacuum.

The theory known as the *Donnan Equilibrium* refers to the ionic equilibrium which is established in the presence of a membrane impermeable to certain non-dialysing electrolytes or miscellæ. The simplest system usually used to demonstrate this principle is a solution of a proteinate, designated NaR, where R represents the non-dialysing radical, on one side of a "non-changing" membrane and a solution of an electrolyte with an ion in common, e.g., NaCl, on the other side of the membrane. The membrane is equally permeable to Na and Cl ion but impermeable to R. Due to the non-diffusibility of R and that at equilibrium the product of Na and Cl ion on both sides of the membrane are equal, the concentration of the Na ion in the compartment containing the NaR originally, will be greater and there is an unequal distribution of this ion on both sides of the membrane and the solution in the NaR compartment will have a higher osmotic pressure than the solution of the other compartment.

From the above discussion it will be seen that the application of physico-chemical concepts to the study of a living organism is circumscribed in that no allowance is made for the continuous adaptation of a living cell to its environment. If this important characteristic is lost sight of, then the conclusions drawn must be erroneous as they cannot be applied to the functioning, living cell.

Carbohydrate Metabolism

Ingested carbohydrate material is split to the hexose stage in the gastro-intestinal tract and resorbed into the hepatic circulation. This process occurs gradually, less than 1.8 gms. of dextrose per kgm. of body weight per hour, reaches the liver. From the liver, the dextrose, which is not utilized by the hepatic glands or stored as glycogen, is passed on to the systemic circulation. In this process the dextrose undergoes a second glycogenesis, the store of glycogen in the muscles being comparable to that in the liver. Ordinarily, tissues, except liver, do not form dextrose from the glycogen stored. Muscle tissue may store sugar as such and give up the stored dextrose to the venous circulation if the blood sugar falls below that of the tissues. Dextrose is readily diffusible and tissues with a large surface of their capillary bed offer little resistence to the diffusion of this sugar. The gradient of diffusion depends upon the ratio — Plasma sugar : Tissue sugar. While the dextrose entering the liver may be either polymerized to glycogen, utilized as such or passed on into the systemic circulation, the dextrose absorbed by the muscle tissues and polymerized to glycogen must undergo esterification with phosphoric acid before it can be utilized. The difference between the dextrose concentration of the arterial blood and the dextrose concentration in the venous blood represents the amount of dextrose absorbed and utilized by the tissues. Glucolysis, the splitting of dextrose to lactic acid also occurs *in vivo*. In every cell, the respiration of which has been disturbed, glucolysis occurs. Cells which are degenerating and losing their nuclei have a very low oxygen intake associated with a high glucolysis. The red blood corpuscles, which have no nuclei, exhibit glucolytic activity. Likewise, the retina removed from the eyes of warm-blooded animals exhibits glucolysis. According to Cori, this is due to a damage to the tissue in the process of removing the retina. It has been stated that the lactic acid content of the aqueous humor exceeds that of the blood and also that glucolysis occurs in the eye tissue *in vivo*, probably mostly in the lens and other non-vascularized eye tissue (see next section).

Circulation, Fluid Exchange and Respiration of the Eye

The vascularized tissue in direct contact with the aqueous chamber is the iris and the ciliary body. The uvea, which is rich

in blood vessels, and borders on the intraocular fluids, maintains an active metabolic exchange with the intraocular fluids. The extent of this exchange must depend upon the state of permeability of the capillary endothelium, upon the metabolism of the uveal cells and on the permeability of the uveal tissue as a whole.

The circulation of the three main divisions of the uvea differs in character. The choroid circulation is predominantly an enormous net of small veins, the ciliary body has small veins which develop directly from the arteries; capillary loops are but rarely seen in this body, and the veins are of a truer character than the choroidal veins which can only be arbitrarily separated from the capillaries. The iris shows the same richness in capillaries as other body tissue. Part of the branches of circulus arteriosus major pass into the iris and run in its anterior layer toward the pupillary margin, part of the branches reach this and part of the branches form by anastomosis a delicate vascular circle, the arteriosus minor, which lies near the marginal edge of the iris. However, tissue cells lie between the aqueous chamber and the capillaries, the fluid dialysing through the tissue cells into the aqueous chamber. The vascular system of the uvea is characterized by its great reducing power. Basic dyes, which circulate in the other blood vessels of the body without reduction, are immediately reduced to the colorless state when they reach the vessels of the iris, ciliary body or choroid. This would indicate the presence of a very active auto - oxidation - reduction system in the eye.

Rate of Circulation of Blood Through the Eye

An average of 1.68 cmm. (1.48 cmm. to 2.02 cmm.) of blood per minute circulates through one vein of the rabbit's eye. Expressed in terms of gms. of tissue this represents 6.61 cmm. of blood per minute circulating through the eye per 100 gms. of tissue. Strong illumination or heat will increase this volume.[12]

The Oxygen Consumption of the Eye

The oxygen consumption of the eye in daylight was determined by Tashiro Kaneko as 4.79 vol. % or 0.275 cmm. per minute.[13]

The total respiration of the eye is very small. It has been calculated that the eye uses 438 mgms. of glucose per day. Part

of this is glucolyzed. That is to say 248 mgms. are glucolyzed (broken down to lactic acid) and 190 mgms. are oxidized. The amount of oxygen available in the eye suffices for the oxidation of 200 mgms. glucose only.[12]

The Rate of Formation of Aqueous Humor

Friedenwald[14] in a recent publication on the circulation of the aqueous states the rate of flow in the dog's eye is 1 cmm. per minute and the total volume of aqueous humor is from 0.3 to 0.5 ccm. At this rate of flow Friedenwald estimates that the fluid is normally replaced every five to eight hours. The replacement of the aqueous within an hour after removal, indicates the increase in permeability due to the paracentesis.

Friedenwald concludes that there is a continuous through-and-through circulation of aqueous within the eye and that the chemical composition of the aqueous is compatible with this view.

Permeability

The degree to which a substance will diffuse from the blood-stream into the tissue field is governed by the permeability of the capillaries, by the degree of dispersion of the substance, by the electric charge on the substance or by its polarity, by its concentration within and without the capillaries and by the hydrostatic pressure of the blood and the oncotic pressure of blood and tissue fluid.

Nervous Control of Capillary Permeability

The permeability of the blood vessels of the eye is under nervous influence. The retinal, choroidal and ciliary arteries have definite vasoconstrictor fibres which run into the cervical sympathetic.

Forces Active in the Formation and Maintenance of the Aqueous Humor

The forces active in the distribution of constituents of the blood, between the blood and the aqueous humor have been variously held to be (A) secretion, or transudation or exudation; (B) dialysis, diffusion; (C) filtration, ultrafiltration. In considering this problem the nature of the barrier must be taken into account. When the problem is approached through the

study of an active metabolite such as glucose, the nutrition of the tissue will play an important part. Even with less active metabolites it is difficult to separate the changes due to the metabolic activity of the tissue from the changes due to physico-chemical forces. Various attempts have been made to account for the formation of the aqueous as a result of hydrostatic forces.

Kronfeld[15] also maintains that there is an intimate relation between intracoular pressure and the amount of aqueous humor in the eye. The amount of inflow, according to Kronfeld, depends mainly upon the capillary blood pressure and on the permeability of the lining membrane. Kronfeld concluded that the secondary aqueous resembles the venous blood while the primary aqueous resembles the arterial blood. He found the increase in protein content in the regenerated aqueous much less in human eyes than in animal eyes. According to Kronfeld the regeneration of the aqueous is a physico-chemical process in which the Donnan equilibrium does not play an important part and which probably completely corresponds neither to dialysation nor to ultrafiltration.

Duke-Elder is the chief exponent of the theory which considers the aqueous humor a dialysate of the blood, modified by the Donnan membrane equilibrium.

In considering the physico-chemical forces which may affect the intraocular pressure he showed that the osmotic concentration of the blood affects the intraocular pressure. Raising the oncotic pressure of the blood by means of intravenous gum arabic injections caused a fall in the intraocular pressure, while lowering the oncotic pressure of the blood by diluting the blood with isotonic saline, caused a rise in the intraocular pressure. The intraocular pressure was also found to fall with an increase in the osmotic concentration of the electrolytes of the blood and to rise with a decrease in the crystalline concentration of the blood.

Duke-Elder's contention, quoted by Yudkin,[16] that the fact of a greater concentration of anions (e.g., Cl) in the aqueous fluid than in the blood, disproves the transudation theory is no longer pertinent to the question. Heubner and Meyer-Bisch[17] determined the sulfates in the aqueous humor of rabbits' eyes and found the concentration of SO_4 ion to lie between 5.8 to

6.4 mgms.%. The concentration of this anion in the ultrafiltrate from the serum of the animals was found to be an average of 10.7 mgms.%. This anion is present in far lesser concentration in the aqueous than in the blood serum. The fact that this radical is a compound, bivalent ion and that membranes are less permeable to bivalent ions than to monovalent ions, must be remembered. Furthermore, the effect of the sulfate radical on colloid systems differs from that of the monovalent chloride ion.

The increase in permeability of the barrier due to repeated paracentesis and consequent increase in the concentration of the larger molecules in the aqueous fluid forms the subject of a paper by Franchetti and Wieland.[18] They determined the normal protein content of the aqueous fluid of the rabbit's eye. The average concentration was 0.05%. The protein concentration of the regenerated aqueous is always higher and the increase is maintained for a week. The protein content of the human aqueous humor was found to vary between 0.019 to 0.034%, the aqueous obtained on second puncture had a higher protein concentration but the increase was not as marked as in the case of rabbits. In acute glaucoma the primary aqueous had a protein content of 0.213 to 0.457% and this was increased in the regenerated aqueous to 0.474 to 0.767%. The greatest increase in protein concentration was found in hydrophthalmus congenitus. In this condition the primary aqueous protein concentration varied between 0.782 to 2.37% while the regenerated aqueous had a protein concentration which varied between 2.86 to 3.2%.

In the controversy as to the forces active in the formation of the aqueous humor, the fluid is usually considered as derived directly from the capillaries. The formation of the aqueous, however, does not take place like the formation of the lymph with the capillary endothelium as the permeable membrane. The "barrier" between blood and aqueous fluid is far more complex, it consists of the capillary endothelium, the iris endothelium and the epithelium of the ciliary body. In the formation of the lymph two factors are mainly concerned; the hydrostatic pressure in the vessels which results in filtration, and osmosis and diffusion. The chemical factor for lymph formation consists in the steady cellular metabolism which results in the removal of substances from the tissue fluid and the addition of metabolites to the tissue fluid, bringing about osmotic differences between tissues and blood.

Filtration and dialysis no doubt are processes active in the formation of the aqueous humor, but they are modified by the nature of the barrier and the constant changes taking place in the barrier itself. All the attempts which have been made to explain the formation of the aqueous humor by one or the other physico-chemical concept illustrate the fallacy of reasoning from the behavior of a single component of a complicated system to that of the whole. The formulation of theories based upon observation of one or even two or three units of a biological system for the modus operandi of the system functioning in the living organism invariably leads to error. Thus, if one compares the concentrations of the electrolytes in the aqueous humor with the concentrations of the electrolytes in the blood plasma one cannot consider that the relationship could be explained by the Donnan equilibrium as might be the case if the concentrations of only one such body are considered. One wonders whether the authors who refer to the Donnan membrane equilibrium have ever attempted to formulate the mathematical equation which would include all the electrolytes present in the blood-aqueous system. The exponents of the theory that the Donnan equilibrium is the deciding factor in the distribution of electrolytes between blood and aqueous have made use of various modifying influences in order to reconcile existing concentrations with those demanded by the theory. The mathematical equation for the Donnan equilibrium does not provide for adsorption, non-solvent space, or compound formation. The introduction of such modifications immediately removes the argument from the domain of pure physical chemistry.

Since it has been shown that the permeability of the blood-aqueous barrier is under nervous influence, it may be said that not only the intraocular pressure but also the composition of the aqueous is under nervous control, though only secondarily.

Summarizing, one may say that the aqueous is derived from the blood circulating in the vessels of the iris and ciliary body. The fluid passes through the capillary endothelium, traverses the cells and tissue fluid, the iris endothelium or ciliary epithelium. In its passage from the blood to the aqueous chamber it is subject to metabolic influences and to storage as glucose and glycogen. Vasomotor processes by increasing or decreasing permeability, as well as hormonal influences on permeability, af-

fect the blood-aqueous relationship. The diffusion of the constituents will also be affected by their relative concentration in blood and aqueous. The circulation and exchange of the aqueous is slow. Since it represents the nutrient medium for the non-vascularized eye tissue it is subject to metabolic changes, due especially to the metabolic activity of the lens. The lens capsule has been shown to be freely permeable to the electrolytes as well as organic metabolites in the aqueous fluid.

PART V

STUDY OF CHANGES IN CONCENTRATION OF DEXTROSE IN AQUEOUS HUMOR AND BLOOD

DANIEL B. KIRBY, M.D., AND RENEE V. E. WIENER, Ph.D.

THE WORK embraced the following: (1) determinations of dextrose content of blood and aqueous humor in cases of cataract in diabetics and non-diabetics and (2) the changes in concentrations as compared to fluctuations in blood sugar level under varying conditions. Normal controls were not available in this series. The examination of the humors of the eyes of laboratory animals has given much information in the past, but there is no way of duplicating human pathological conditions in the animal eye. Of the seventy-five patients who submitted to this test a total of 130 times, no harm resulted in any case. The technique must be perfect. In only two cases was there bleeding from the iris and this absorbed promptly. In no case was there retinal hemorrhage or exudate dependent on the paracentesis even though it was performed in the presence of hemorrhages and exudates in the retina of certain diabetics. In one case the cataracts present progressed to complete opacity rapidly after the test but it is believed that this would have occurred in any event as they were of the rapidly progressive anterior and posterior subcapsular variety.

Yudkin and associates[19] analyzed the aqueous humor of the dog. Obtained under local anasthesia it had an average dextrose concentration of 77 mgms. per 100 ccm., while that obtained under amytal anasthesia had a slightly higher concentration. Since the fasting blood sugar of the dog normally varies from 70 to 100 mgms. per 100 ccm. the aqueous sugar concentration lies slightly below that of the blood.

Duke-Elder determined the dextrose concentrations in the arterial blood plasma, venous blood plasma and primary and secondary aqueous humor of three rabbits. He found but slight variation between primary and secondary aqueous humors. The

average dextrose concentration of the primary aqueous humor was 159 mgms. per 100 ccm., while the average dextrose concentration of the arterial plasma was 148 mgms.[20]

The relation of the blood and aqueous dextrose during a sugar tolerance test was determined in two rabbits. In the rabbit which had a normal blood sugar concentration before the administration of dextrose the blood sugar, following the administration of 1.7 gms. of dextrose in 10% solution, rose 108% above its fasting value during a period of two hours. The increase in dextrose concentration of the primary aqueous humor during the first hour of the test was 35%. The increase in concentration of dextrose found on second paracentesis was 52% for the left eye, taken during the first eighty-five minutes of the test and 54% for the right eye, taken during the second hour of the test.

Bellavica[21] determined the blood and aqueous sugar of animals under normal conditions and also under the influence of pilocarpin, atropin, dionin, mercuric bichloride and insulin. No matter how the blood sugar was affected its concentration always was higher than the concentration of the aqueous humor sugar.

The dextrose concentration of the lens exceeds that of the aqueous humor or of the vitreous. O'Brien and Salit[22] determined the chemical constituents of enucleated cattle eyes and report the aqueous dextrose concentration 73.1 to 87.1 mgms. per 100 ccm., that of the vitreous an average of 55.5 mgms. and that of the lens an average of 134.7 mgms.

Walter Dieter determined the sugar concentrations of the blood, primary and secondary aqueous humors of a case of diabetic coma. Primary aqueous humor dextrose, 293 mgms. per 100 ccm.; venous blood sugar, 289 mgms.; secondary aqueous humor dextrose, 301 mgms., and venous blood sugar, at the time, 306 mgms.[23]

Cohen and associates[24] in their work on the ocular fluids and blood of dogs determined the sugar and lactic acid concentrations of the blood and combined ocular fluids during hypoglycæmia due to repeated doses of insulin. While there is a certain parallelism between the decrease in dextrose concentration of the blood and combind ocular fluid this does not appear to be the case for the lactic acid concentration. The dextrose concentration of the blood greatly exceeds that of the combined ocular

fluids. The lactic acid concentration of the blood was found to be about one-third that of the combined ocular fluids in the pre-insulin state, after repeated doses of insulin the blood lactic acid exceeded that of the aqueous and vitreous. In this connection the increase in lactic acid due to muscular contractions which raises the blood lactic acid concentration even after a slight exercise, as well as the possibility that part of the lactic acid of the combined ocular fluids may have resulted from glucolysis after the enucleation of the eye, must be taken into account.

The lactic acid content of the blood and aqueous humor was determined by Wittgenstein and Gaedertz.[25] In the absence of anti-glycolytic agents, such as sodium fluoride, the lactic acid content of the aqueous humor was found to increase from an original concentration of 22 mgms. per 100 ccm., to 33 mgms. in forty-five minutes. If the lactic acid content of the blood was raised by muscular work the lactic acid of the aqueous increased likewise, but the increase was less rapid and less drastic than the increase in the blood. There also seemed to be a lag in the subsequent removal of the lactic acid from the aqueous humor as compared to the blood.

Little is known about glucolysis in the eye *in vivo*, at least by direct experiment. It has been stated that this form of breakdown takes place in the non-vascularized tissue, e.g., the lens. It also takes place in the retina.

In the study of the relationship between dextrose concentration of the blood and dextrose content of aqueous humor, the physico-chemical forces active in the distribution of the substance as well as biological factors must be taken into account. A perfect comparison could only be made were it possible to withdraw, simultaneously, blood from the vessels of the uvea and aqueous fluid from the anterior chamber. The dextrose in its passage from the capillaries through the uveal tissue into the aqueous chamber is subjected to metabolic influences. The choroid is said to have an intense sugar metabolism and according to Takeushi contains an oxidase and a reductase. The ciliary body tissue undoubtedly has similar properties. Whatever the nature of the sugar metabolism in the eye, the differences between blood and aqueous sugar concentrations seem to be well established. Friedenwald[26] who studied the permeability of the lens to crystalloids found it freely permeable to dextrose. In view of the

reported concentrations of dextrose in the lens and aqueous humor, one may feel tempted to postulate directional permeability of the lens capsule for this metabolite. According to Fischer[27] the lesser concentration of dextrose in the aqueous humor is mainly due to the metabolic activity of the lens.

The factors which influence the concentration of dextrose in the aqueous humor are: (A) the concentration of dextrose in the blood plasma, (B) the permeability of the blood vessels, (C) the permeability of the uveal tissue barrier, (D) the rate of diffusion of dextrose, (E) the fluid balance between blood and tissues, (F) the dextrose metabolism in vascularized eye tissue, (G) the dextrose metabolism in non-vascularized eye tissue, including glucolysis and (H) the rate of circulation of the aqueous.

The dextrose concentrations of aqueous humor, capillary and venous blood were determined on seventy-five patients. Twenty-three of these cases were diabetics and nineteen of these had incipient or immature cataracts. Twenty-five cases were non-diabetics and twenty-one of these had cataracts. These forty-eight cases were examined and tested in the afternoon clinic and the patient, after one hour's rest following the paracentesis, was allowed to go home. The other series of twenty-seven patients were hospitalized and the dextrose determinations made under varying conditions. In ten of these cases both the primary and secondary aqueous humor dextrose concentrations were compared with the blood dextrose concentrations.

Paracentesis of the anterior chamber need not be feared if proper precautions are observed. According to Burbank tapping of the aqueous of the eye was done by the ancient Arabians as shown by the picture of a syringe used for this purpose in an incunabula. Graves reported his method of puncture with a sharp cataract knife and aspiration into a tube. Most of the investigators who have used human aqueous in their work obtained it by aspiration with a syringe. Local anasthesia is necessary and a speculum to support the lids. Fixation forceps without catch are employed to steady the globe. Fixation is best taken in the upper nasal quadrant and the syringe (tuberculin 1 ccm.) with a 27-gauge needle entered horizontally opposite the fixation. If the syringe and needle are rotated in a drilling fashion, the needle is introduced without even dimpling the

cornea, without undue force or sudden jump into the anterior chamber. The method is painless. After paracentesis, a light dressing is applied. This is removed in twenty-four to forty-eight hours. At this time the anterior chamber has returned to its previous condition. In a few cases there were subconjunctival hemorrhages at the site of fixation. In one case of old iritis there was a flaring up of the process for a few days Otherwise, the post-operative course of these patients was uneventful.

Table I gives the dextrose concentration of blood and aqueous humor in a group of twenty-five non-diabetic patients. The average age was 56.8 years. The average blood dextrose was 120 mgms. per 100 ccm., the average dextrose content of the aqueous humor varied from 103 mgms., making the ratio of aqueous to blood 0.85. The dextrose value of the aqueous humor varied from 135 mgms. to 60 mgms.; it exceeded that of the blood in only two cases.

In Table II the dextrose concentrations of aqueous humor and blood of twenty-three diabetic patients are shown. The highest dextrose value of the aqueous humor in this group was 186 mgms., the lowest 93 mgms. per 100 ccm. The average blood sugar value was 146 mgms., that of the aqueous 124 mgms., making the ratio of aqueous to blood 0.85. The average age was 58.8 years. Both groups were examined and tested during the afternoon clinic, one to three hours after their mid-day meal.

Twenty-seven patients were hospitalized and the value of the aqueous humor dextrose in relation to the blood sugar concentrations determined. Thirteen of these had dextrose tolerance tests. Three had insulin depletion tests and in six cases the rise of the dextrose concentration following food intake was determined on the primary aqueous humor. In ten cases the dextrose concentrations of the primary as well as of the regenerated aqueous humor were determined, in two of these cases after food and insulin, in five cases after food intake, and in three cases during sugar tolerance tests.

The patients were hospitalized on the day prior to the test. The fluid intake and output during sugar tolerance test periods were noted. The urine was routinely examined. A somewhat modified Hagedorn-Jensen method was used for the determination of the dextrose on the filtrate obtained by the Somogyi

TABLE I — *Comparative Blood and Aqueous Dextrose Tests*
Twenty-Five Non-Diabetic Cases

			MGMS. DEXTROSE PER 100 CCM.					
CASE NO.	SEX	AGE	BLOOD	CORRECTED FOR FILTRATE VOLUME	AQUEOUS	RATIO	EXAMINATION *of* EYES	COMPLICATIONS
231138	M	57	83	89	60	.67	Cataract vacuoles. Lamellar separation.	Hypertension $\frac{170}{110}$
229596	F	75	95	102	93	.91	Cataract incipient. Water-split sutures. Lamellar separation. Diffuse lens opacities.	Aortic insufficiency. Aneurysm.
279387	F	62	99	106	119	1.12	Cataract incipient. Lamellar separation. Water-split sutures.	Art. sclerosis.
291202	M	46	108	125	81	.65	Cataract anterior posterior subcapsular opactities.	
229530	M	59	121 109	129 117	113 90	.87 .76	Cataract cortical.	Art. sclerosis.
280659			113 115	121 123	90 96	.74 .78	Cataract posterior cortical subcapsular vacuoles.	
286181	M	45	115	123	86	.69	Cataract nuclear. Narrow vessels. Fundus hazy. Pigmented spot in macular region.	
282464	F	57	116	124	88	.72	Cataract cortical.	Art. sclerosis. Hypertension $\frac{180}{80}$
264765	M	34	119	127	127	1.00	Primary optic nerve atrophy. No lens opacity.	Lues. Nephrectomy.
290987	M	40	107 131	114 140	82 88	.71 .62	Cataract anterior and posterior subcapsular opacities. Myopia.	
276428	M	33	127 105	136 112	120 86	.88 .76	Secondary glaucoma.	Wass. 4 plus.

Case No.	Sex	Age	MGMS. DEXTROSE PER 100 CCM. Blood	Corrected for Filtrate Volume	Aqueous	Ratio	Examination of Eyes	Complications
276889	F	69	119 115	127 123	88 80	.69 .65	Cataract incipient. Water-split sutures.	Art. scler. Cardiac hypertrophy. Hypertension $\frac{188}{110}$
258817	F	68	129 113	138 121	117 93	.84 .76	Cataract Lamellar separation. Water-split sutures.	Neuritis.
287899	M	66	122 150	131 160	98 135	.74 .84	Dense opacity in the vitreous complicated posterior cortical cataract.	Varicose veins. Art. sclerosis. Arthritis.
285861			120	128	98	.76	Cataract cortico-nuclear opacity. Lamellar separation. Old water-split sutures.	Myocarditis.
228873	F	83	120	128	92	.71	Cataract nuclear and posterior cortical opacity. Presbyopia and myopia.	Hypertension $\frac{200}{120}$ Obesity.
278962	F	65	127	136	91	.66	Cataract incipient opacity—O.S.	Art. sclerosis. Hypertension $\frac{170}{90}$
229732	F	70	125	134	88	.65	Cataract—Old water-split sutures and diffuse opacity.	Art. sclerosis.
247197	F	54	121	129	117	.91	Cataract retinal hemorrhages. Old scars in macula.	
277302	F	63	127	136	125	.92	Iridocylitis.	Chronic tonsillitis.
290881	M	36	129	138	102	.73	Cataract mature senile cortical.	Hypertension $\frac{148}{90}$
206741	M	49	130	139	127	.91	Cataract subcapsular. Chronic glaucoma.	Hypertension $\frac{160}{100}$
190981	M	58	131	140	123	.87	Cataract incipient vacuoles. Water-split sutures.	Art. sclerosis. Cardiac hypertrophy.
289212	F	67	137	147	88	.59	Optic nerve atrophy. No lens opacities.	
291112	M	30	164	175	122	.68		

method for de-proteinization. Lithium citrate was used as anti-coagulent. It was determined that lithium citrate in the proportions used did not affect the ferri-cyanide of the reagent. A 10% dextrose solution was used in the sugar tolerance tests, in the proportion of 0.8 gms. dextrose per kilogram of body weight. This is sufficient to obtain the maximum rise and return to normal within the period of the test which was from two to two and one-half hours.

Table III gives the results of sugar tolerance tests in thirteen cases. In five of these the aqueous was first removed before the dextrose ingestion and the aqueous of the fellow eye one hour after the dextrose ingestion. In the remaining cases the first aqueous removal was from forty to ninety minutes after the dextrose ingestion and comparison was made with the dextrose concentration of the aqueous humor of the other eye during the last period of the test.

The average fasting concentration of dextrose in the primary aqueous humor was 88 mgms. per 100 ccm., varying from 103 mgms. to 80 mgms. The primary aqueous humor of the other eye, at the first hour period showed an average concentration of 109 mgms. per 100 ccm. (120 mgms. to 92 mgms.). The increase in dextrose concentration was considerably less in the aqueous humor than in the blood. The difference in dextrose concentration between capillary blood and primary aqueous humor increased from an average of 21 mgms. per 100 ccm. in the fasting conditions to a difference of 85 mgms. per 100 ccm. one hour after the ingestion of the dextrose. Thus the increase in the primary aqueous humor dextrose is far less than in either venous or capillary blood. The average increases, based on the fasting values were: Aqueous humor 24%, capillary blood 80%, and venous blood 65%. This relation between dextrose of aqueous humor and dextrose of the blood was usually maintained in the non-diabetic type of curve as well as in the diabetic type (see Fig. I, showing composite curves of both types and average aqueous dextrose concentrations). In Fig. II the curves obtained on Case 4, Table I, are shown. They are representative of the non-diabetic group.

In the remaining eight cases the changes in dextrose concentration of the primary aqueous humors were determined during the second period of the test. In the first four of these cases

TABLE II — *Comparative Blood and Aqueous Dextrose Tests*
Twenty-Three Cases of Known Diabetes Mellitus

			MGMS. DEXTROSE PER 100 CCM.					
				Corrected for Filtrate				
Case No.	Sex	Age	Blood	Volume	Aqueous	Ratio	Examination of Eyes	Complications
291879	M	64	82	88	93	1.05	Old superf. opacities in cornea. Vitreous clear.	Chronic tonsillitis.
283136	M	58	103	110	93	.83	Cataract. Vacuoles and water-split sutures.	Art. sclerosis. Hypertension $\frac{160}{90}$
280947	F	59	109	116	102	.87	Pigmented spots in macular region. Many small hemorrhages.	Hypertension $\frac{210}{110}$
171451	F	57	104 146	111 156	101 119	.90 .79	Cataract incipient.	General art. sclerosis. Hypertension $\frac{150}{85}$
260689	F	63	121 134	129 143	100 109	.77 .76	Cataract incipient. Vacuoles.	Hypertension $\frac{160}{100}$ Otitis media.
69300	M	66	123	132	121	.91	Cataract incipient, peripheral water split sutures, opaque nucleus.	Art. sclerosis. Chronic myocarditis.
233194	F	45	121	129	98	.75	Cataract—cortical.	Hypertension $\frac{160}{80}$ T.B. Cavities—old.
189241	F	68	122 161	130 173	93 95	.71 .54	Cataract incipient. Water-split sutures. Vacuoles.	Hypertension $\frac{190}{70}$ Otitis media.
173805	F	64	133	142	123	.85	Cataract secondary to old uveal inflammation.	Hypertension $\frac{260}{110}$
288117	M	55	138 128 130	148 137 140	100 100 115	.67 .74 .82	Cataract—cortical.	Obesity. Art. sclerosis.

TABLE II — Concluded

CASE NO.	SEX	AGE	MGMS. DEXTROSE PER 100 CCM. BLOOD	CORRECTED FOR FILTRATE VOLUME	AQUEOUS	RATIO	EXAMINATION of EYES	COMPLICATIONS
83393	F	54	137	146	130	.89	Cataract—cortical.	
275500	F	67	134	143	97	.68	Cataract cortical, traumatic O.D. Incipient senile O.S.	Hypertension $\frac{165}{80}$
174028	M	63	123 134	132 143	123 143	.93 1.00	Cataract Lamellar separation, sutures, vacuoles.	Art. sclerosis.
272660	F	58	133	142	129	.90	Retinitis. Macular lesions.	Tabies dorsalis Wass. 4 plus.
230191	M	49	141	151	128	.84	Cataract—cortical.	
85322	F	56	142	152	123	.81	Cataract peripheral opacities.	Chronic arthritis.
223978	F	48	144	154	139	.90	Cornea, lens vitreous clear. Art. sclerosis.	Hypertension $\frac{182}{95}$
231140	F	58	143	153	132	.86	Cataract. Diffuse opacities. Few vacuoles.	Varicose veins.
250340	M	57	150 150	160 160	101 130	.63 .81	Cataract immature, O.D. Incipient O.S. Water-split sutures, vacuoles, hemorrhages.	Impetigo.
280527	F	73	156	167	112	.67	Cataract. Water-split sutures. Lamellar separation. Incipient senile cortical cataract.	General Art. sclerosis. Tertiary syphilis.
217068	F	69	180 205	192 219	149 183	.76 .83	Cataract cortico-nuclear.	Art. sclerosis.
268898	F	52	211	216	166	.76	Cataract antero posterior subcapsular.	Art. sclerosis. Aortitis.
235314	M	50	222	226	186	.82	Cataract posterior cortical.	Art. sclerosis. Stricture of male urethra.

TABLE III—*Sugar Tolerance Tests. Dextrose Concentrations of Capillary, Venous Blood and Aqueous Humor.*

CASE NO.	NO., AGE AND SEX OF PATIENT	MGMS. DEXT. PER 100CCM. AQUEOUS					MGMS. DEXTROSE PER 100CCM. OF BLOOD						CARBOHYDRATE TOLERANCE AND NATURE OF CURVES	EYE CONDITION	COMPLICATIONS
							CAPILLARY BLOOD AT TIME OF AQUEOUS			VENOUS BLOOD AT TIME OF AQUEOUS					
		First Taken	First Eye	Minutes Interval	Second Eye	Difference	I	II	Diff.	I	II	Diff.			
1.	204743 53 yrs. Male.	Fasting	85.-	65	93.-	8.-	150.-	180.-	30.-	120.-	200.-	80.-	Lag type curve. Tissue metab. slightly decreased. Steep ascent venous c.	Incipient, senile, cortical cataract, sclerosis.	Slight bradycardia and hypertension.
2.	291281 32 yrs. Female	Fasting	80.-	60	92.-	12.-	102.-	175.-	73.-				Normal.		
3.	280947 58 yrs. Female.	Fasting	81.-	60	120.-	39.-	81.-	246.-	165.-	90.-	180.-	90.-	Diabetic type curve. Normal absorption, decreased oxidation.	Nuclear cataract, split sutures, many small hemorrhages.	Diabetes Mellitus, mild. Hypertension 210/110
4.	229596 76 yrs. Female.	Fasting	103.-	65	118.-	15.-	132.-	188.-	56.-	125.-	172.-	47.-	Normal old age curve.	Incipient cataract. Water-split sutures. Lamellar separation.	None.
5.	185880 56 yrs. Female.	Fasting	90.-	55	120.-	30.-	110.-	180.-	70.-	105.-	165.-	60.-	Acicular type, steep ascent and return.	Chronic glaucoma.	Hypertension 195/105. Chronic nephritis.
6.	258817 70 yrs. Female.	60 min. post dextr.	82.-	70	79.-	—3.-	165.-	102.-	—63.-	160.-	110.-	—50.-	Normal old age curve. Tissue metab. slightly decreased.	Old water-split sutures and lamellar separation.	None.
7.	287899 71 yrs. Male.	60 min. post dextr.	90.-	60	104.-	14.-	204.-	154.-	—50.-	207.-	144.-	—63.-	Slight decrease in Tolerance and in Tissue Metabolism.	Post cortical cataract. Dense vitreous opacities.	Varicose Veins.
8.	280659 59 yrs. Female.	60 min. post dextr.	97.-	60	103.-	6.-	150.-	127.-	—23.-				Normal curve.	Cataract.	
9.	277068 71 yrs. Female.	60 min. post dextr.	163.-	60	186.-	23.-	253.-	223.-	—30.-	248.-	230.-	—18.-	COH tolerance decreased. Diabetic type curve. Tissue met. decreased.	Double aphakia.	Moderate diabetes Mellitus. Arterio-sclerosis.
10.	228732 72 yrs. Female.	60 min. post dextr.	167.-	60	178.-	11.-	265.-	270.-	5.-	230.-	252.-	18.-	Diabetic type curve. Normal tissue metabolism.	Incipient cataract. Lens opacities.	Arterio-sclerosis. Mild hypertension.
11.	296722 50 yrs. Female.	90 min. post dextr.	110.-	60	121.-	11.-	209.-	195.-	—14.-				Diabetic type curve. Much decreased tissue met. with normal absorp.	Cataract.	Renal calculus.
12.	247297 54 yrs. Female.	60 min. post dextr.	112.-	60	122.-	10.-	167.-	155.-	—12.-	152.-	140.-	—12.-	Lag type curve.	Cataract.	None.
13.	206741 49 yrs. Male	40 min. post dextr.	130.-	60	142.-	12.-	205.-	215.-	10.-	194.-	166.-	—28.-	Diabetic type curve. Delayed absorption by tissues with normal ox.	Extracted cataract O.S. Incipient cataract O.D.	Diabetes Mellitus.

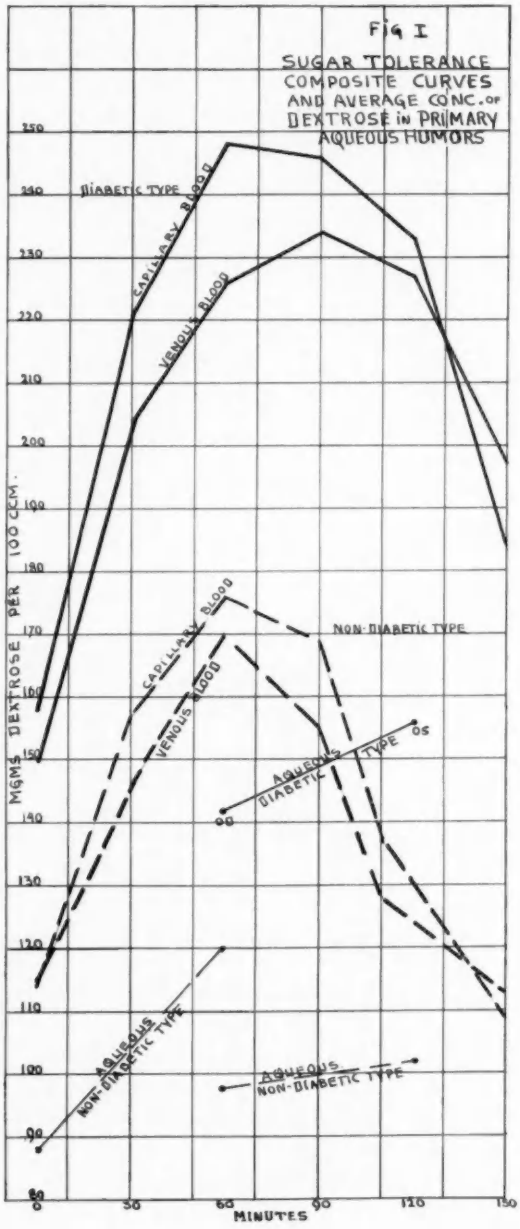
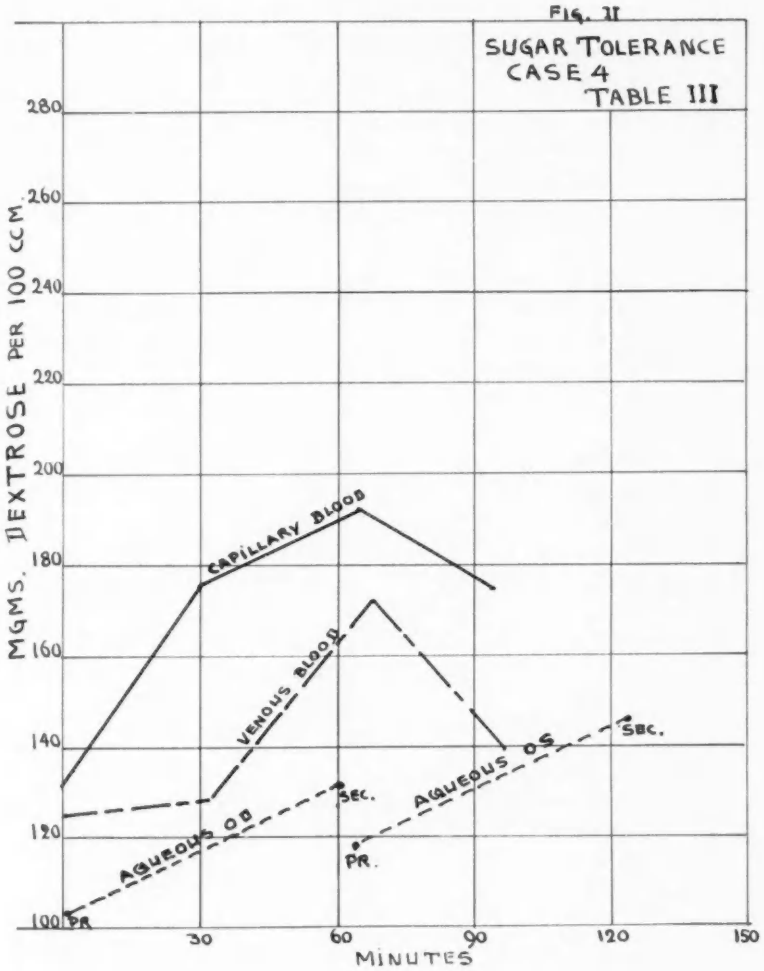


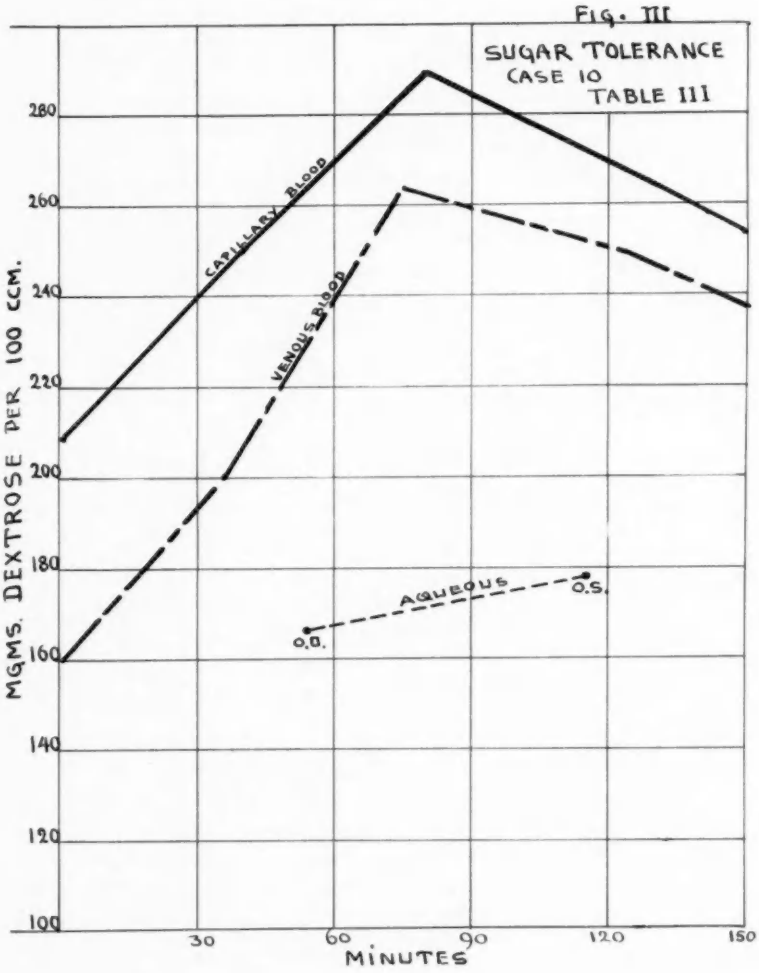
Fig I
SUGAR TOLERANCE
COMPOSITE CURVES
AND AVERAGE CONC. OF
DEXTROSE IN PRIMARY
AQUEOUS HUMORS
DIABETIC TYPE
CAPILLARY BLOOD
VENOUS BLOOD
NON-DIABETIC TYPE
CAPILLARY BLOOD
VENOUS BLOOD
AQUEOUS DIABETIC TYPE OS
AQUEOUS NON-DIABETIC TYPE
AQUEOUS NON-DIABETIC TYPE
MGMS DEXTROSE PER 100 CCM.
MINUTES
250
240
230
220
210
200
190
180
170
160
150
140
130
120
110
100
90
80
0
30
60
90
120
150

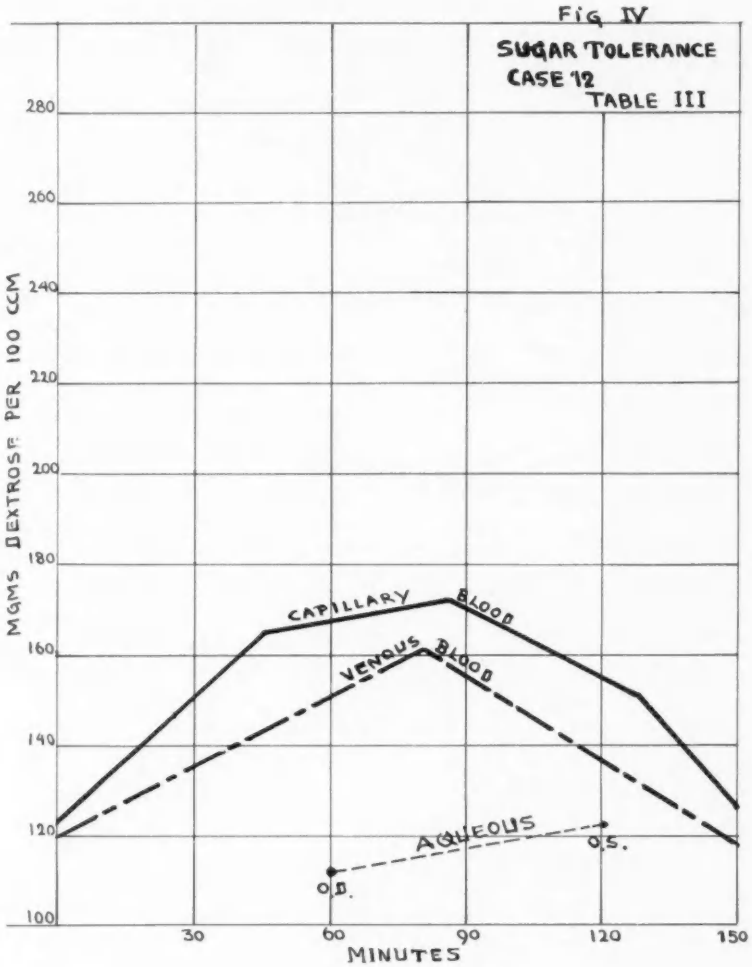


(Nos. 6, 7, 8, and 9), the peak of the blood sugar curve had either been passed or was passed during the first third of this period. The changes in the primary aqueous humor dextrose concentrations were, therefore, determined during the descending part of the blood sugar curve. With the exception of Case No. 6 (258817) the dextrose concentrations of the primary aqueous humors increased while the blood dextrose decreased. The average increase in the primary aqueous humor was 10 mgms. dextrose per 100 ccm. of fluid, the average decrease in the blood sugar

during this period was 40 mgms. per 100 ccm. for capillary blood and 44 mgms. per 100 ccm. for venous blood.

In Cases Nos. 10, 11, 12, and 13, the period of the aqueous humor dextrose determinations embraces the flat portion of a prolonged blood sugar curve. Fig. III gives the curves and aqueous humor dextrose on Case 10, diabetic type and Fig. IV shows the curves obtained on Case 12, non diabetic type. In all these cases there was a plateau effect with the peak in the middle of the period. The average increase in dextrose concen-





trations of these four cases was 11 mgms. per 100 ccm. of aqueous humor. The blood dextrose at the two points showed but little change. In two cases there was a slight decrease, 13 mgms. per 100 ccm., in two cases a slight increase, average of 7 mgms. per 100 ccm.

There is a definite delay or lag in the rise of dextrose concentrations of the primary aqueous humors as compared to the increase in blood sugar values. It is retarded and prolonged as compared to the rise and fall of the blood sugar. It is rather

significant that of the thirteen cases examined, only two showed a normal fluid economy (Nos. 2 and 6). These two cases were also the nearest to an assumed normal tolerance curve. Five of the cases were diabetics and the average rise in aqueous humor dextrose for these five cases was 19 mgms. as compared to an average rise of 11 mgms. for the eight non-diabetic patients.

The most marked increases in the primary aqueous humor dextrose concentrations observed were:

48% above its fasting value in a cataract case with diabetes and hypertension.
33% above its fasting value in a case of glaucoma with nephritis and hypertension.
23% above its first hour value in a case of double aphakia, mild diabetes mellitus and arterio-sclerosis.

The first of these cases excreted 25% of the fluid intake while the other two cases were unable to void any urine during the test period.

The dextrose depletion (insulin reaction) tests are shown on Table IV. The first case, a woman of forty-seven years with diabetes of sixteen years' duration, showed an increase in the primary aqueous humor dextrose although there was a considerable drop in the blood dextrose. The difference between the primary aqueous humor dextrose concentration and the capillary blood sugar, two hours after a carbohydrate intake of 40 gms. and fifty-five minutes after 25 units of insulin was 207 mgms. per 100 ccm. This difference decreased in the next hour to 60 mgms. per 100 ccm. In this connection the differences between capillary and venous blood sugar concentrations should be noted. The first post insulin difference is 80 mgms., the second is 42 mgms. This patient had been on insulin treatment for nine years. Her present daily dosage is 35 units (25 — 0 — 10). The ability of her tissue to remove dextrose from the arterial capillary circulation is above normal. Both the low value of the primary aqueous dextrose of the right eye as compared with the blood sugar at the time, as well as the rise in aqueous dextrose when the blood sugar was decreasing are interesting features in this case.

The second case is that of a woman of sixty-three years, cataracts, diabetes mellitus, hypertension and severe pyorrhea. Before insulin injection the difference in dextrose concentration

TABLE IV — *Dextrose Depletion — Insulin Reaction Tests.*
Dextrose Concentrations of Capillary, Venous Blood and Aqueous Humor.

Case No.	No., Age and Sex of Patient	Mgms. Dextrose per 100ccm. Aqueous Humor					Mgms. Dextrose per 100ccm. Blood						Eye Conditions	Complications
							Capillary blood at time aqueous humor			Venous blood at time aqueous humor				
		First Taken	First Eye	Minutes Interval	Second Eye	Difference	I	II	D.ff.	I	II	D.ff.		
1.	A. S. 47 yrs. Female.	120 min. post 50 COH, 55. min. post 25 U. Ins.	153.-	60	188.-	35.-	360.-	250.-	—110.-	280.-	208.-	—72.-	Incip.ent, peripheral cataract, many smal hemorrh., split sutures.	Diabetes Mellitus. 16 yrs. duration.
2.	336159 63 yrs. Female.	Ante Insulin 180 min. post 60 COH, 2nd Eye post 10 U. Ins. (50 min.)	232.-	60	262.-	30.-	290.-	250.-	—40.-				O.D. Severe cortical catar act. Incipient cataract O.S.	Diab. Mell. 5 yrs. Hypertension. Pyorrhea.
3.	335672 26 yrs. Male.	First aq. ante food or treatment Second post 1 hr. infusion and ins.	182.-	90	168.-	—14.-	265.-	178.-	—87.-				None.	Diabetes Mellitus. Hospitalized for Acidosis.

between primary aqueous humor and blood was 58 mgms. per 100 ccm. On hour after 10 units of insulin the conditions were reversed. The concentration of the dextrose in the primary aqueous humor of the other eye exceeded that of the blood by 12 mgms. The changes in permeability due to infection may have played a part, severe pyorrhea may exert a decided effect on tissue permeability.

The third case is that of a young male diabetic of twenty-six, hospitalized for acidosis, with negative eye condition. Ex-

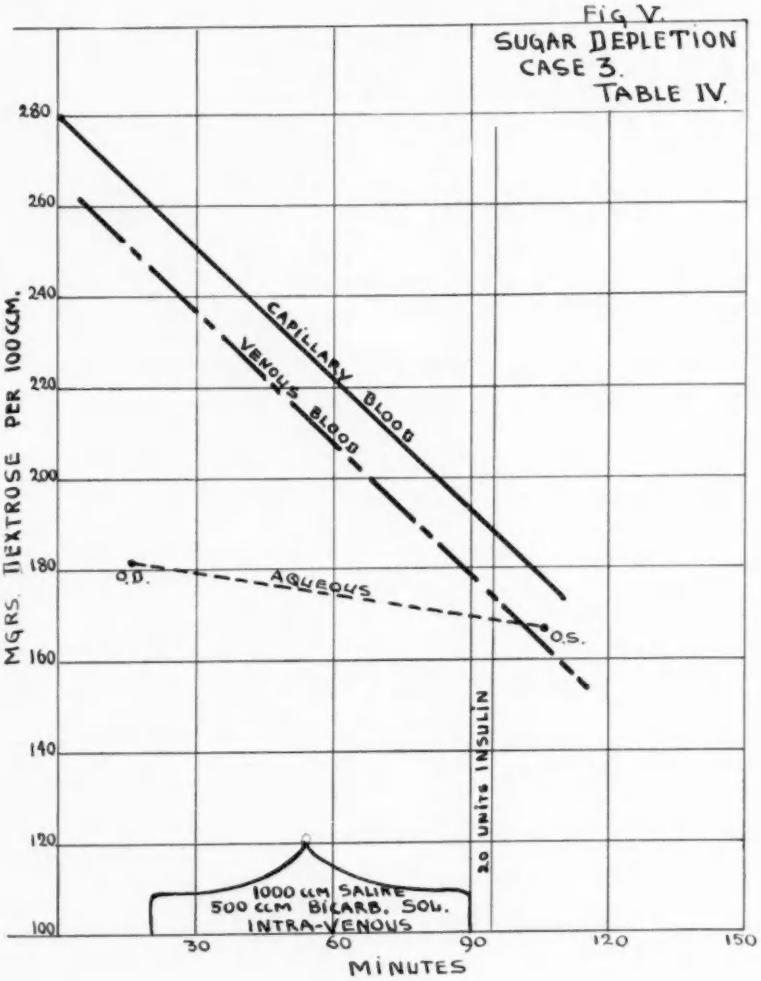


TABLE V — *Dextrose Concentrations in Blood and Aqueous Humor. After Food Intake.*

No.	No., Age and Sex of Patient	Mgms. Dext. per 100ccm. Aqueous Humor					Mgms. Dextrose per 100ccm. of Blood						EYE CONDITION	COMPLICATIONS
							Capillary blood at time aqueous humor			Venous blood at time aqueous humor				
		Conditions Under Which First Aqueous Was Taken	First Eye	Minutes Interval	Second Eye	Difference	I	II	Diff.	I	II	Diff.		
1.	289023 63 yrs. Female.	80 minutes post 65 gms. COH	119.-	65	137.-	18.-	160.-	148.-	—12.-	148.-	138.-	—10.-	Retinal changes. Lens haze. Nuclear sclerosis, old exudates and edema.	Very mild diabetes mellitus. Arterio-sclerosis.
2.	199535 56 yrs. Female.	90 min. post 127 gms. COH and 200 min. post 50 U. Insulin	160.-	125	155.-	—5.-	190.-	192.-	2.-	185.-	178.-	—7.-	Negative.	Diabetes mellitus (3 yrs.) Hospitalized for mod. acidosis.
3.	302364 73 yrs. Female.	115 minutes post 100 gms. COH	201.-	65	168.-	—33.-	228.-	180.-	—48.-	214.-	166.-	—48.-	Senile cortical cataract.	Arterio-sclerosis.
4.	318410 52 yrs. Male.	100 minutes post 80 gms. COH	116.- (O.D.)	60	89.- (O.S.)	—27.-	155.-	160.-	5.-	147.-	157.-	10.-	Senile, cortico-nuclear cataract O.S., prel'y iridectomy 2 ms. ago. OD normal, sl. amber hue.	None.
5.	251158 60 yrs. Male.	90 minutes post 90 gms. COH	93.-	65	88.-	—5.-	128.-	114.-	—14.-	127.-	110.-	—17.-	Incipient cataract, lamellar sep. water-split sutures, 1 in each eye.	Mild hypertension.
6.	195549 68 yrs. Male.	85 minutes post 80 gms. COH	90.-	80	113.-	23.-	150.-	170.-	20.-	120.-	130.-	10.-	Cataracts. Lamellar separation and water-split sutures.	Arterio-sclerosis and hypertension. Vericose veins.

cept for low, intraocular tension this case resembled the first one quoted. The ante-insulin, non-fasting difference between blood and aqueous humor dextrose was materially decreased during a ninety-minute interval. During this time the patient received a slow infusion of saline and bicarbonate, a total of 1500 ccm. and shortly before the second determinations 20 units of insulin. The decrease in dextrose concentration in the primary aqueous humor was very slight compared to that in the blood. See Fig. V.

In Table V the comparisons made between dextrose concentrations of primary aqueous humor and blood after food are shown. The first case is that of a mild diabetic with senile cataract which showed a slight increase in primary aqueous dextrose concentration during the later part of the test period, when the capillary and venous blood sugar concentrations were decreasing. This patient had a general arterio-sclerosis and evidence of this condition in the eye. The difference in dextrose concentration between capillary and venous blood is very slight. Whether this is due to a general decrease in permeability of the blood vessels or decrease in ability of the tissues to remove and metabolize the sugar, or both, cannot be differentiated. The rise in the secondary aqueous of the right eye (see section on Table VI) indicated that the secondary aqueous dextrose may be influenced by the sugar content of the uveal tissues rather than the blood or that the dextrose content of the secondary aqueous is an expression of the blood dextrose content at the time of the removal of the primary aqueous. After paracentesis the anterior chamber fills rather quickly and if the capillaries dilate properly and the tissues are normally permeable the crystalloids of the plasma will pass without hindrance.

The second case of this series is a woman, hospitalized for a mild acidotic condition. The test was performed ninety minutes after 64 gms. of carbohydrate intake and two hundred minutes after 50 units of insulin. Before food intake and insulin injection the fasting blood sugar was 176 mgms. per 100 ccm. of capillary blood, and 174 mgms. for the venous blood. The large dose of insulin held the blood sugar concentrations at practically "fasting" level during a period of almost four hours despite the ingestion of a total of 127 gms. of carbohydrate material. There is but a slight change in the dextrose concentration of the primary aqueous which decreases 5 mgms. per 100 ccm., or 3% of

its original concentration. This change is within the experimental error of the analytical method.

The third case, a woman of seventy-three, had a distinctly decreased carbohydrate tolerance although not classified as diabetic. Both venous and capillary blood show an abnormal, diabetic-like rise in dextrose concentrations following the carbohydrate meal. The primary aqueous humor dextrose concentration, determined during the descent of the blood sugar, almost parallels it.

The fourth case is that of a man of forty-two, with a cortico-nuclear cataract in the left eye, while the right eye is normal except for a slight amber hue of the lens, indicating pathological sclerosis of the nucleus. A preliminary iridectomy had been performed on the left eye two months ago. The drop in the dextrose concentration in the primary aqueous humor of the two eyes is most probably due to a difference in permeability between the two eyes. This case is further discussed in the next section (Table VI).

The fifth case had a normal carbohydrate metabolism as shown by the blood sugar concentrations two hours after a light breakfast; 103 mgms. per 100 ccm. of capillary blood and 99 mgms. in venous blood. The drop in the primary aqueous concentration is very slight, from 93 mgms. per 100 ccm. of fluid to 88 mgms. The minimal rise in the blood dextrose, coupled with the negligible difference between capillary and venous blood sugar concentrations indicates a very slow entrance of dextrose into the systemic circulation. This may either be due to a decreased resorption from the intestinal tract or to an increased destruction of the sugar by intestinal bacteria. The removal of dextrose from the blood on its passage from the arterial to the venous circulation on part of the tissues is below normal.

The last case had a carbohydrate metabolism normally retarded for her age. The rise in the primary aqueous dextrose concentration might lead one to believe that a peak had occurred in the blood sugar curve during the interval between the first and second aqueous humor.

In all these cases the character of the food was taken into account. Due to the dextrose being derived from starch (the lunch consisted mainly of vegetables, potatoes, bread and butter) the resorption of dextrose was much slower than in sugar tolerance tests.

TABLE VI—*Dextrose Concentrations in Primary and Secondary (Regenerated) Aqueous Humor Capillary and Venous Blood*

Case No.	No., Age and Sex of Patient	Mgms. Dextrose per 100ccm. Aqueous Humor						Mgms. Dextrose per 100ccm. of Blood								EYE CONDITION	COMPLICATIONS
		Conditions Under Which Aq. Was Taken	Right Eye			Conditions Under Which Aq. Was Taken	Left Eye			Capillary Blood at Time of Aqueous Humor				Venous Blood at Time of Aqueous Humor					
		Time and Food	I	Min. Int.	II	*Time and Food*	I	Min. Int.	II	O.D. I	O.D. II	O.S. I	O.S. II	O.D. I	O.D. II	O.S. I	O.S. II		
1.	289023 63 yrs. Female.	320 min. p. bfst., 80 m. post lunch.	119.-	60	127.-	145 min. post lunch.	137.-			160.-	148.-	147.-		148.-	139.-	147.-		Retinal changes, lens haze. Nuclear sclerosis. Old exudates and edema.	Very mild diab. mell. Arterio-sclerosis.
2.	336159 63 yrs. Fema'e.	140 min. p. food and ante Insulin. 10 U.	232.-	60 **	288.-	205 min. p. food and 55 min. post Ins.	262.-	55	226.-	290.-	254.-	250.-	195.-					Dense cortical cataract O. D. Incipient Cataract O.S.	Diab. mell. 5 yrs. Hypertension. Pyorrhea.
3.	J. R. 68 yrs. Female.	Fasting. Sec'y 55 m. Post dextrose.	85.-	55	120.-					128.-	212.-			120.-	185.-			Acute Glaucoma and Iritis.	Gave diabetic-like response to tolerance Test.
3'.	Same	120 min. p. bfst. and sec. post dextrose.	78.-	60	114.-					160.-	223.-			147.-	215.-				
4.	199535 56 yrs. Female.	90 min. post lunch & 50 U. Insulin.	160.-	135	161.-	230 min. post 50 U. Insulin.	155.-			190.-	192.-	192.-		185.-	178.-	178.-		None.	Diab. mell. Hospitalized for mod. acidosis.
5.	302364 73 yrs. Female.	115 min. p. 100 gms. COH.	201.-	60	173.-	130 min. p. 100 gms. COH.	168.-			228.-	185.-	180.-		214.-	170.-	166.-		Senile cortical cataract, water-split sutures.	Arterio-sclerosis diabetic like respone to COH intake.
6.	318410 52 yrs. Male.	100 min. p. 80 gms. COH.	116.-	60	168.-	165 min. p. 80 gms. COH.	89.	55	76.-	155.-	160.-	160.-	140.-	147.-	156.-	157.-		Senile cortico-nucl. cataract. O.S. O.D. normal, lt. amber hue.	None except slight enlargement of prostate.
7.	251158 60 yrs.	90 min. p. 90 gms. COH.	93.-	60	86.-	155 min. p. 90 gms. COH.	88.-	50	90.-	128.-	115.-	113.-	100.-	127.-	111.-	109.-		Inc. senile cataract lamellar separation one water-split suture in each eye.	Mild hypertension.
8.	312621 50 yrs. Female.	Ante tolerance Test. Sec'y 60 min. p. dextr.	108.-	60	136.-					134.-	237.-			120.-	200.-			Mature, senile cataract O.D. (O.S. extracted)	
9.	J. H. 74 yrs. Female.	Ante tolerance Sec'y 55 min. p. dextr.	176.-	55	210.-	60 min. post Dextrose.	169.-	55	174.-	186.-	275.-	279.-	309.-	190.-	275.-	282.-	305.-	Double Aphakia. O. D. extracted, O.S. extraction followed by discission	Diabetes mell. Arterio-Scler. Myocarditis.
10.	195549 68 yrs. Female.	85 min. post 80 gms. COH.	90.-	75	118.-	165 min. p. 80 gms. COH.	113.-			150.-	169.-	170.-		120.-	130.-	130.-		Cataract. Lamellar separation, water-split sutures.	Arterio-sclerosis. and mild hypertension.

** (50 minutes post Insulin)

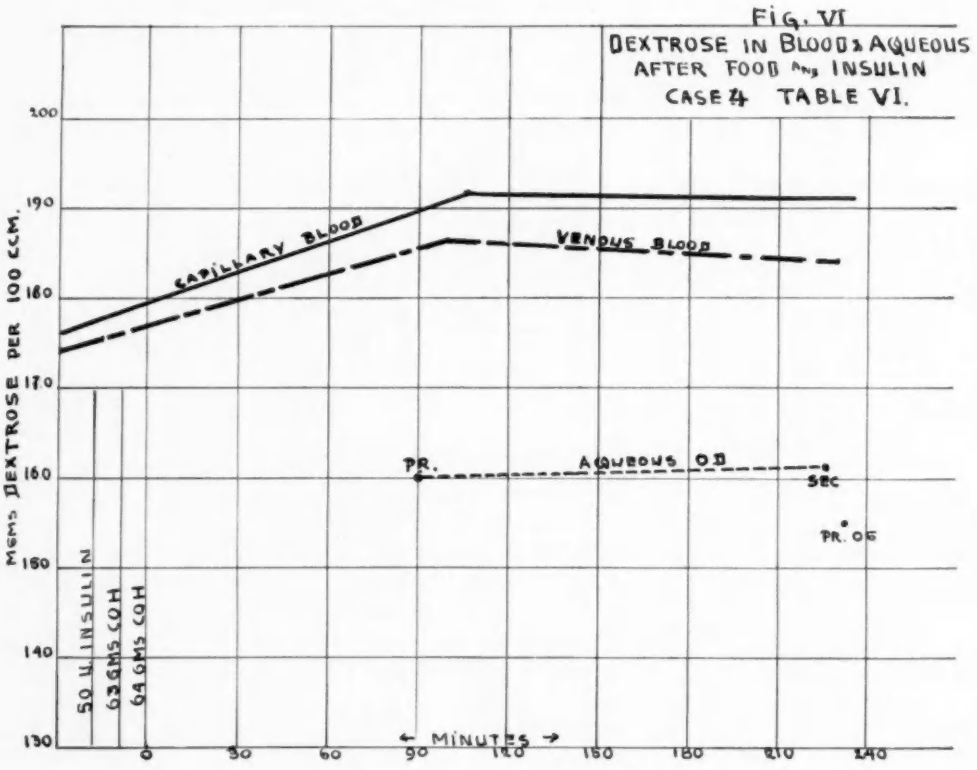
In Table VI the relation of dextrose concentration in the primary and regenerated aqueous humor to that of the blood is shown.

The first is that of a mild diabetic with arterio-sclerosis. The diabetic condition is controlled by a slightly restricted diet without insulin. The determinations on the primary and secondary aqueous fall in the descending part of the blood sugar curve. The increase in dextrose concentration of the regenerated aqueous humor is very slight and the concentration lies below that of the primary aqueous of the other eye taken immediately afterwards. This may possibly mean a decrease in capillary permeability.

The second case is that of a woman of sixty-three, with moderately severe diabetes mellitus of five years' duration, on insulin treatment. The dextrose concentration of the capillary blood and primary aqueous of right eye was determined 140, resp. 150 minutes after breakfast before the morning dose of 10 units of insulin. It was again determined fifty minutes after the insulin injection. During this period the capillary blood sugar concentration fell from an initial concentration of 290 mgms. per 100 ccm. to 254 mgms. or suffered a loss of 12.5% of the original concentration. In contrast to the drop in blood sugar concentration there is a rise in the aqueous humor dextrose concentration. This is much more marked in the regenerated aqueous of the right eye than in the primary aqueous of the left eye, obtained five minutes later. The dextrose concentration in the regenerated aqueous humor was 288 mgms. per 100 ccm., practically the same as the pre-insulin blood sugar level. This can be understood if one takes into account (a) the rapid regeneration of the aqueous humor and (b) the interval between subcutaneous insulin injections and drop in blood sugar, which for doses of this strength lies between fifteen to twenty minutes. Since the first aqueous was taken ten minutes before the insulin injection, there was ample time for the anterior chamber to fill before the insulin had any effect on the blood sugar. The dextrose concentration of the primary aqueous humor of the other eye, obtained five minutes later, was 262 mgms. per 100 ccm. It lies slightly above the capillary blood sugar level. This negative difference between capillary blood sugar and aqueous humor dextrose is still further increased in the regenerated aqueous humor of this eye, determined sixty minutes later; again

illustrating the relatively slow removal of dextrose from the aqueous humor, the rapid regeneration of the aqueous humor and the increased permeability after paracentesis.

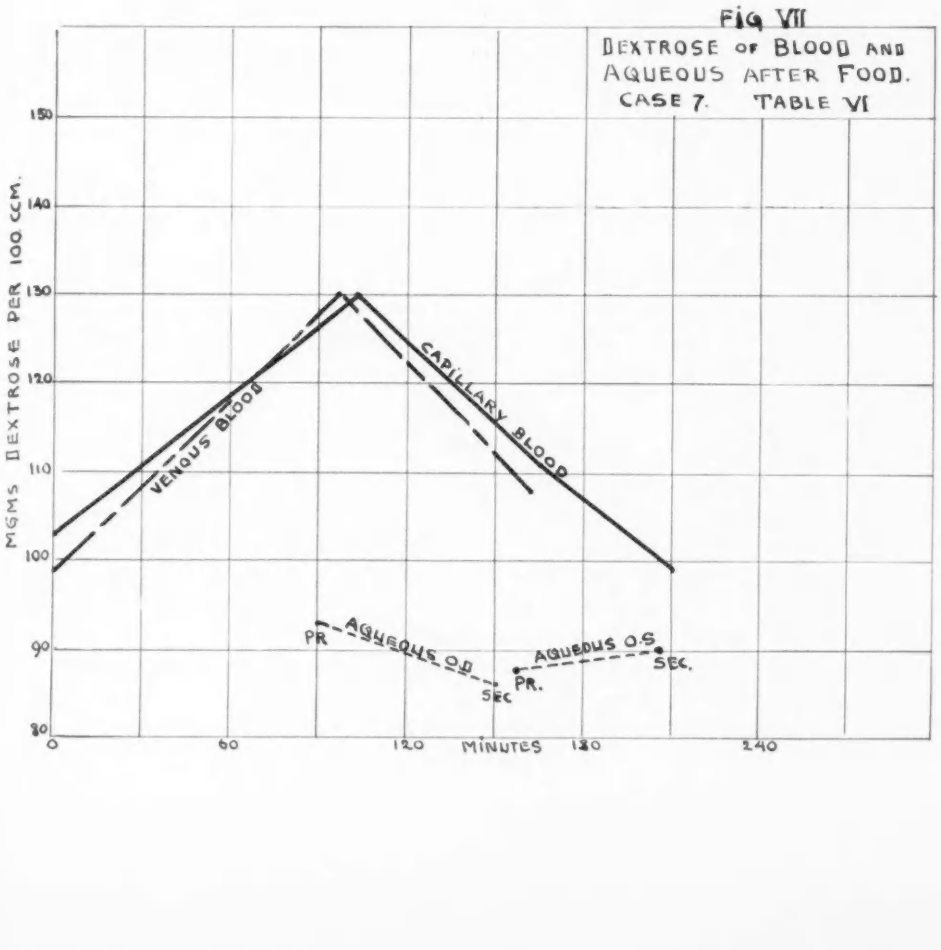
Case No. 3 illustrates a decrease in aqueous humor dextrose in the presence of inflammation. This case, a woman of sixty-eight with acute glaucoma and iritis, gave a diabetic-like response to dextrose ingestion. The fasting blood sugar, 128 mgms. per 100 ccm. of capillary blood (120 mgms. for venous blood), increased following the ingestion of 50 gms. of dextrose in 10% solution to 212 mgms. resp. 185 mgms. in one hour. During this time the dextrose concentration of the aqueous humor of the same eye increased from a fasting level of 85 mgms. per 100 ccm. to 120 mgms. The increase in dextrose concentration of the aqueous humor determined in a second tolerance test on the same patient, two hours after breakfast, resembled that of the first tolerance.



The fourth case (199533) discussed with Table V showed no change in secondary aqueous humor dextrose concentration as compared to the dextrose level of primary aqueous humor. This is accounted for by the unchanged concentration in blood sugar during the period of the test and most likely by a corresponding unchanging concentration of dextrose in the uveal tissues (See Fig. VI).

Case No. 5 showed a decreased dextrose concentration of the secondary aqueous humor. It will be noted that the first aqueous was withdrawn practically two hours after a 100 gms. carbohydrate meal and that the blood sugar was decreasing. The secondary aqueous by reflecting this drop in blood and uveal tissue dextrose may indicate decreased capillary permeability and absence of the usual increase following paracentesis.

Case 6, also discussed in connection with Table V, had a unilateral cataract with the right eye normal except for the



amber hue of the lens nucleus. In the "good" eye there was an increase in the secondary aqueous humor dextrose concentration, which rose above that of the capillary or venous blood sugar level. This may be taken to indicate a peak in the blood sugar curve occurring during the time intervening between the two determinations. The aqueous humor of the cataractous eye had a primary dextrose concentration far below that of the capillary or venous blood. The secondary aqueous humor of this eye showed a further decrease in dextrose concentration. Both the determinations of the cataractous eye were made late in the curve during which time the capillary blood sugar showed a sharp drop. However, the difference in dextrose concentrations in the two eyes is striking and may point to a decreased permeability of the uveal tissue and probable deficiency in nutrient material in the aqueous humor.

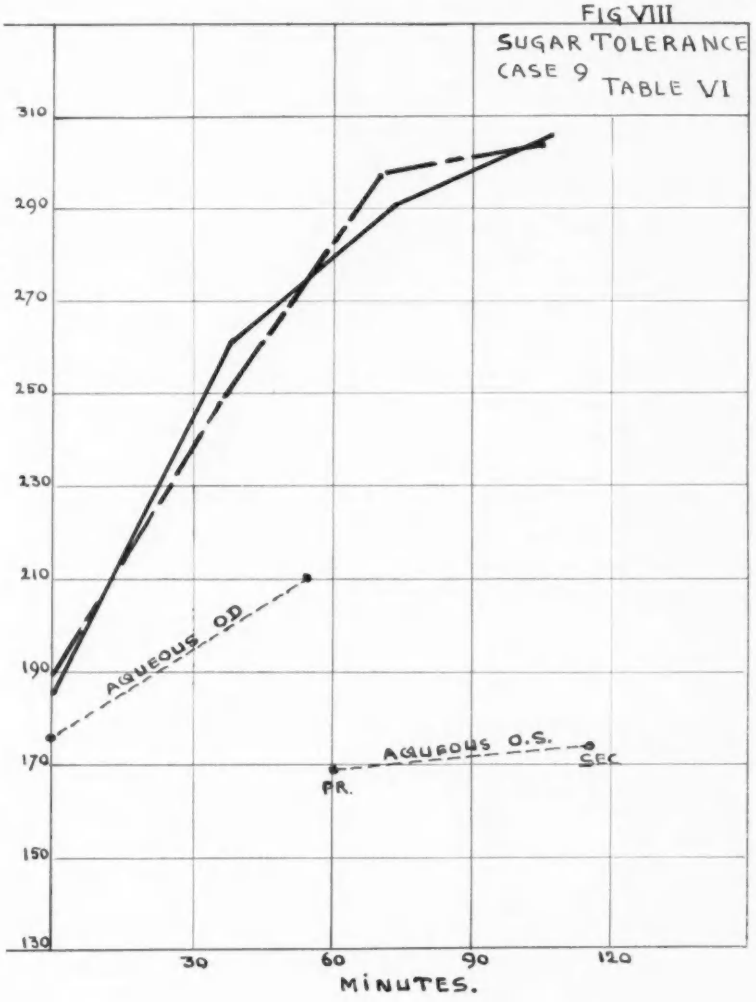
Case 7 could also be interpreted on the basis of decreased permeability. The dextrose concentrations of the aqueous humors of both eyes are at the low fasting level, though the first aqueous was obtained ninety minutes after food. The concentration is practically stationary in the secondary as well as in the primary aqueous (Fig. VII).

In Case 8 the fasting blood sugar was above normal and the response to dextrose ingestion was increased. The capillary blood sugar doubled its value in forty minutes after food intake. The curve was of the acicular type (see Part II). The fasting aqueous sugar value is 10% below that of the blood. The increase in dextrose concentration of the secondary aqueous was 26% above that of its fasting value (136 mgms.), although the capillary blood sugar at the time (237 mgms.) was still 64% above its fasting level.

Case 9, a diabetic woman of seventy-four, has a different dextrose concentration in the aqueous humor of the two eyes. Both eyes are aphakic, the right by capsulotomy extraction, the left by capsulotomy extraction followed by discission by the Wheeler method. The dextrose concentration of the right eye taken in the fasting condition was above that of the left eye, taken sixty minutes after the ingestion of dextrose in a sugar tolerance test. The rise in the dextrose concentration of the secondary aqueous humor of the right eye, while lagging behind, yet reflects the rising blood sugar, but the rise in the secondary

aqueous of the left eye, also determined during the ascending part of the blood sugar curve, is so slight that it may be said to be absent. The lower concentrations in the left eye may have an interpretation in the absence of the posterior capsule barrier, the hyaloid membrane, which had been divided by discission while the right was intact (Fig. VIII).

The last case is also discussed in connection with Table V. The glucose concentration of the secondary aqueous reflected the rise in blood sugar.



Discussion

None of these patients were in the normal class as far as their eyes were concerned and a large percentage had a deficient carbohydrate metabolism. Any inferences which are made are taken to apply only to the cases under consideration.

In general it has been found that the aqueous humor dextrose concentration does not equal that of the blood and does not change parallel to the blood. To explain the lower concentrations of the sugar in the aqueous especially as compared to the non-fasting blood one may postulate: (a) The uveal tissue acts as a barrier to the rapid diffusion of the dextrose, (b) Metabolism of the sugar as it passes through the uveal tissue, (c) Slow rate of filtration of fluid from the capillary bed through the uveal tissue into the anterior chamber, (d) Sugar bound to the plasma proteins and not diffusing into the chamber, (e) Rapid use of sugar in aqueous and vitreous, (f) Differences in dextrose content of uveal capillaries as compared to either finger-tip blood or venous blood, (g) The presence of unknown reducing substances in the blood not present in the aqueous humor.

The uveal tissue cells and interstitial fluid may be considered the semi-permeable membrane which acts as the barrier to the rapid entrance of the dextrose into the aqueous. The increase in blood sugar following carbohydrate ingestion may result in increased concentration of dextrose in the uveal tissue fluid and ciliary epithelial cells. The dextrose may be present either as such or stored as glycogen. Part of the glycogen may remain stored, while part is utilized in metabolic processes. The rate of diffusion of the sugar from the uveal tissues into the aqueous humor may depend largely upon its concentration both in the uveal tissue and in the aqueous humor. The dextrose of the aqueous humor is used by the lens and cornea and also leaves the anterior chamber through the Schlemm's canal. It may also be partly resorbed by the iris and possibly by the ciliary body. Part of it also diffuses through the vitreous and is metabolized by the retina. There may also be spontaneous glucolysis in the aqueous and in the vitreous. When the blood sugar falls very rapidly, as after insulin injection, the aqueous humor dextrose may remain for a while elevated above the blood sugar level. The changes in relationship between the blood and aqueous humor dextrose may be considered a gradual change towards equi-

librium on the two sides of a semi-permeable membrane interposed between the capillary blood and the anterior chamber. The rate of this change towards equilibrium may be modified by the state of the permeability of the barrier in the individual case. After the blood sugar has been stationary for a long period, as after fasting, the aqueous dextrose has a concentration of 80 to 90% of that of the blood dextrose. Following the ingestion of carbohydrate material the increase in concentration of dextrose in the aqueous humor lags behind that of the blood. Furthermore, the blood sugar may reach its peak and start diminishing, but as long as the blood sugar level is above that of aqueous humor, dextrose will continue to diffuse in. In diabetes mellitus the tissue has partly lost its ability to metabolize glucose and the concentration of dextrose in the blood and in the uveal tissues is above normal in these cases. Hence, relatively more dextrose should diffuse into the aqueous humor and at a relatively more rapid rate. A more rapid rate of increase could not be demonstarted, but in the diabetics examined the aqueous humor dextrose was increased, both in the fasting state and after carbohydrate intake.

While the increase in sugar concentration of the blood is probably the most outstanding feature in diabetes mellitus there are other changes in blood constituents which may have a bearing. The fats, fatty acids, lipides and sterols of the blood are often increased. The serum proteins as well as the calcium of the blood are usually a high normal in uncomplicated diabetes-mellitus.[28] In diabetes mellitus there are changes in cellular and membrane permeability and further a definite change from the normal in the water metabolism of the body.

A loss in tissue permeability is generally found in old age.[29] Likewise, there are also changes in permeability and water economy of the aging, non-diabetic eye. Changes in permeability, such as, for instance, brought about by sclerosis of the vessels, interfere with the diffusion of metabolites into the tissue fluid and aqueous humor.

The regenerated aqueous humor, under normal conditions of dilatation of the capillaries and permeability of uveal tissue, represents the inflow of plasma dialysate during the interval following closely upon the first paracentesis. If, during the interval between the two paracenteses, there is a decided drop in

blood sugar, such as may occur after insulin injection in a diabetic, the dextrose concentration of the secondary aqueous humor will more closely approximate the blood sugar level at the time or shortly after the first paracentesis than at the time of the second. This will then result in an increased concentration of dextrose in the aqueous as compared to the blood.

Summary of Part V

The dextrose content of the primary aqueous humor, of the regenerated aqueous humor as well as the blood sugar level of the capillary and venous blood, were determined in a series of seventy-five patients, under varying conditions.

The technique for the paracentesis and withdrawal of the aqueous humor, successfully performed 130 times, is given.

In the first two series the tests were performed in the afternoon clinic. The first of these series consisted of a group of twenty-five non-diabetic patients with cataract. The second of these series consisted of a group of twenty-three patients, diabetics with cataract. The following averages were determined:

Non-Diabetic Group—Average age, 56.8 years; average dextrose concentration of primary aqueous humor, 103 mgms. per 100 ccm.; capillary blood sugar, 120 mgms.; ratio of aqueous dextrose to capillary blood, 0.86.

Diabetic Group—Average age, 58.8 years; average dextrose concentration of aqueous humor, 124 mgms. per 100 ccm.; capillary blood sugar, 146 mgms.; ratio of aqueous dextrose concentration to blood, 0.85.

A third group of twenty-seven patients was hospitalized. The dextrose content of the aqueous humor in its relation to the blood sugar was determined during sugar tolerance tests, after food intake and after insulin. The dextrose concentration in the regenerated aqueous humor was similarly studied in ten of these cases.

The average fasting dextrose concentration in five sugar tolerance tests was 88 mgms. per 100 ccm. and this increased to 109 mgms. during the first hour of the test. The increase in sugar of the capillary and venous blood at that time greatly exceeded

that of the aqueous humor. When the changes in dextrose content of the primary aqueous humor were determined during the second hour of the tolerance tests, it was found that while the blood sugar remained at a certain level or started to fall, the dextrose concentration of the aqueous humor continued to rise. A definite lag in the entrance of the dextrose into the aqueous humor was demonstrated. While the gradient is from the blood to the aqueous the dextrose of the aqueous humor may continue to increase in concentration.

During a rapid fall in blood sugar as after insulin injection the value of the aqueous humor dextrose may be found to exceed that of the blood.

After food the rise in concentration of the dextrose of blood as well as aqueous humor is slower than after dextrose ingestion. In cases of prolonged elevation of blood dextrose there results a prolonged elevation of aqueous dextrose and a relatively higher concentration.

The study of the dextrose concentrations of the regenerated aqueous humor in relation to the blood showed irregularities in behavior of the eyes of certain patients which could only be interpreted on the basis of decreased permeability of the eye tissues. There were also variations in comparing the eyes of the same individual. These could be explained by differing conditions of the two eyes. An hour was usually allowed to elapse between primary and secondary paracentesis. It may prove advantageous to withdraw the secondary aqueous ten to twenty minutes after the primary paracentesis, that is shortly after its reformation.

PART VI.

A STUDY OF THE EFFECT OF CHANGES IN THE NUTRIENT MEDIUM ON LENS EPITHELIUM. CULTIVATED *"IN VITRO"*

1. *Dextrose (glucose), Laevulose (fructose) and Galactose.*

2. *Acetone and Beta-hydroxy-butyric acid.*

DANIEL B. KIRBY, M.D., KEITH ESTEY, AND R. VON E. WIENER

THE GROWTH of a tissue culture depends upon the viability of the explant and the conditions prevailing in the culture medium which is composed of embryonic extract, blood plasma and Tyrode or any similar solution of crystalloids. Study of the metabolism of tissues *"in vitro"* is aided by additions to or variations in the composition of the culture medium.

If, however, the observed results of such a study are used as a correlary for the behavior of tissues under like conditions in the living organism, certain qualifying characteristics of cells growing *"in vitro"* have to be taken into account: (1) The increase in size of a tissue cultured *"in vitro"* takes place by proliferation with or without enlargement of the cells or increase in intracellular material. (2) If the plasma clot method of culture be employed the cells are not provided with a constantly-changing fluid nutrient medium as are the cells in the living body where a freshly-oxygenated nutrient medium is constantly supplying the cells with the necessary salts and organic substances in more or less controlled dilute concentrations and where the products of metabolism are constantly removed. The tissue culture cell is provided with fresh nutrient medium only every forty-eight hours. The waste products of cellular metabolism are allowed to accumulate before being removed. (3) The tension of the

coagulated plasma which forms the scaffolding for the growth of the culture is of influence on the direction form and rate of the growth of the culture.[30] (4) The tissue employed in the culture usually is derived from the embryo body, which in its metabolism (especially that of dextrose) differs from adult tissue. (5) The trauma of the process of transplanting the cells provides a stimulus to growth. (6) The cells live under far more primitive conditions than they do in the living body; and (7) The influences to which the cells, as part of the living organism, are exposed are lacking, e.g., nerve control, hormones and certain enzymes.

As in the living body the cells of a tissue culture derive their energy mainly from the metabolism of glucose. However, actively growing embryonic or regenerating tissue has a relatively higher rate of glucolysis. Krontowski[31] has shown that an actively growing fibroblast culture will utilize as much as five times its dry weight of glucose in twenty-four hours and that about 40% of the glucose utilized is broken down to lactic acid.

Lens epithelium although extremely fragile and transparent has been successfully cultivated by two of us.[32 33] From the aspect of limited oxygen supply and relatively stagnant nutrient medium, lens epithelium "*in vivo*" differs less from lens epithelium grown "*in vitro*" than do other body tissues, under the two conditions. Lens epithelium has been used in this study of the effect of changes in the nutrient medium on its growth. The purpose of this paper is to report (1) the effect of changes in dextrose (glucose) concentration in the nutrient medium as well as the substitution of other sugars (lævulose and galactose). (2) The effect of the two ketone bodies, acetone and beta-oxy-butyric acid, in increasing concentrations in the nutrient medium.

The great variation in the growth of lens tissue even in normal medium (i.e., 50% plasma, and 50% extract) makes it necessary to choose only perfectly healthy, actively-growing cultures of uniform size and type of growth. If a culture was not large enough to divide, a similar culture of the same age and apparent condition was used for control. If eight lens cultures in excellent condition were not available it was not considered worth while to try an experimental medium.

The sugars used were Pfanstiel's preparations. They were added to the experimental Tyrode solution, which, in the case of the galactose and fructose experiments contained no glucose.

This solution was also used for the dilution of the plasma and extract for the experimental cultures. Whenever the plasma and extract were diluted in the experimental cultures, a like dilution was made in the controls with normal Tyrode solution.

For the ketone body experiments a sterile solution of beta-hydroxy-butyric acid and acetone in Tyrode solution was prepared. One ccm. of this concentrated solution contained 10 mgms. acetone and 30 mgms. butyric acid, maintaining the average proportions to each other in which these two ketone bodies appear in the blood. From it the experimental Tyrode solutions were prepared.

In a series of carefully controlled experiments increasing amounts of dextrose concentrations in the experimental medium were used (Table 1). No real difference in growth was noticed until the rather high concentration of 578 mgms. per 100 ccm. of medium (increase of 424 mgms. over the control medium) was reached. In this experiment (No. 50) the controls were decidedly better even after the first forty-eight hours. The experimental cultures became progressively poorer and after two transfers in the same medium (a total life of seven days) all had died. Controls were still in good condition though very thin and irregular in growth, owing to the very dilute medium. In the next experiment the increase to 861 mgms. per 100 ccm. (733 mgms. over the controls) killed the lens epithelium in the first

TABLE 1—*Effect of Increased Concentrations of Glucose*

EXPERIMENT NO.	CONCENTRATIONS OF SUGARS IN MGMS. PER 100 CCM. *Control*	*Experiment*	*Difference*	NO. OF TRANSFERS IN SAME MEDIUM	APPEARANCE OF EXPERIMENTAL CULTURES AS COMPARED TO CONTROLS	*Remarks*
16	166.-	182.-	16.-	3	No difference.	
18	166.-	199.-	33.-	2	No difference.	
38	155.-	299.-	144.-	2	No difference.	
40	154.-	329.-	174.-	2	No difference.	
42	154.-	379.-	225.-	2	No difference.	
44	154.-	428.-	275.-	4	No difference.	Extract diluted 50%
48	154.-	478.-	324.-	2	No difference.	
50	154.-	578.-	424.-	3	Slight inhibition of growth after 48 hrs. Tissue dead after 3rd transfer.	
53	128.-	861.-	733.-	1	Very slight growth in exp. culture in first 48 hrs. Dead after one transfer in same medium.	Plasma and Extract diluted 50%

TABLE 2—*Effect of Fructose in Increasing Concentrations*

EXPERIMENT NO.	CONCENTRATIONS OF SUGARS IN MGMS. PER 100 CCM. *Control* DEXTROSE	*Experiment* DEXTROSE	*Experiment* LÆVULOSE	NO. OF TRANSFERS IN SAME MEDIUM	APPEARANCE OF EXPERIMENTAL CULTURES AS COMPARED TO CONTROLS	*Remarks*
30	158.-	124.-	100.-	3	No difference.	
58	128.-	65.-	800.-	2	No difference.	Plasma and Extract diluted 50%
61	128.-	65.-	1070.-	2	Slight retardation of growth.	
63	128.-	65.-	1300.-	2	Experiments grew poorly. Slight toxicity.	
65	81.-	32.-	1660.-	0	This is toxic. Experimental cultures dead after 48 hrs.	Plasma and Extract diluted 1 to 3.

TABLE 3—*Effect of Galactose in Increasing Concentrations*

EXPERIMENT NO.	CONCENTRATIONS OF SUGARS IN MGMS. PER 100 CCM. *Control* DEXTROSE	*Experiment* DEXTROSE	*Experiment* GALACTOSE	NO. OF TRANSFERS IN SAME MEDIUM	APPEARANCE OF EXPERIMENTAL CULTURES AS COMPARED TO CONTROLS	*Remarks*
24	166.-	132.-	100.-	2	No difference.	
66	166.-	132.-	167.-	3	Slight retardation at first. Growth was totally stopped after third transfer.	
68	128.-	65.-	333.-	2	Poor growth. Practically stopped after 2nd transfer.	Plasma and Extract diluted 50%
71	128.-	65.-	533.-	....	Toxic. Tissue dead in 48 hours.	

TABLE 4—*Effect of Decrease in Dextrose Concentrations*

EXPERIMENT NO.	CONCENTRATIONS OF DEXTROSE IN MGMS. PER 100 CCM. *Control*	*Experiment*	*Difference*	NO. OF TRANSFERS IN SAME MEDIUM	APPEARANCE OF EXPERIMENTAL CULTURES AS COMPARED TO CONTROLS	*Remarks*
22	158.-	124.-	34.-	2	No difference.	
27	153.-	90.-	63.-	2	No difference.	Extract diluted 1 to 10.
34	105.-	12.-	93.-	1	No difference in first 48 hrs. after first transfer controls slightly larger.	Plasma and Extract diluted 1 to 10.

forty-eight hours except for a few straggling cells in two of the cultures. The experiment was repeated on three more cultures in excellent condition. All died within the first forty-eight hours.

After a preliminary experiment in which the lævulose replaced the dextrose in the experimental Tyrode solution with no difference in the cultures, the lævulose was increased to 800 mgms. per 100 ccm. in the experimental medium, without any adverse effect on the cultures. One set of these (two experiments and two controls) was grown on Maximow slides the other set on Carrel slides. In the Maximow slide cultures the controls were better than the experimental cultures, but after one washing and transfer forty-eight hours later to Carrel slides they all grew equally well. In the next experiment (No. 61) the concentration of lævulose in the experimental medium was 1,070 mgms. per 100 ccm. A slight retardation of growth was noted. In the next experiment (No. 63) the lævulose concentration was increased to 1,300 mgms. per 100 ccm. While this concentration was not actually toxic, it retarded the growth which was very poor after the second transfer to the same medium. The toxic concentration was only reached when the experimental medium contained 1,660 mgms. per 100 ccm.

This sugar proved more toxic than glucose (see Table 3). A concentration 333 mgms. of galactose in the experimental medium with approximately 60 mgms. of dextrose present in the medium, inhibited growth after two transfers, while a concentration of 533 mgms. per 100 ccm. killed the tissue within the first forty-eight hours.

The decrease was obtained by diluting the plasma and extract with Tyrode solution from which the glucose had been omitted. In this manner a final difference of 93 mgms. per 100 ccm. of medium between experiments and controls was obtained. In experiment No. 34 the plasma and extract were diluted 1:10 with Tyrode. The experimental medium had a dextrose concentration of 12 mgms. per 100 ccm. Still the experimental cultures did well and no difference was noted after forty-eight hours of growth.

Discussion of Sugar Experiments

The control media used in these experiments contained from 105 to 166 mgms. glucose per 100 gms. The experimental media

contained from 12 to 861 mgms. dextrose per 100 gms. To obtain a satisfactory experimental medium with only 12 mgms. dextrose per 100 gms., it was necessary to dilute greatly the plasma and embryonic extract but still the growth of the experimental culture under these conditions was as satisfactory as that of the control cultures in the diluted medium with normal glucose content. Moderate changes in glucose content had no appreciable effect on the lens epithelium. It was only when the concentration was increased to 578 mgms. and later to 861 mgms. of glucose per 100 gms. that toxic changes due to the concentration of the dextrose were observed. If one may draw a conclusion from this, it is that lens epithelium itself in the living body can withstand tremendous changes in the dextrose concentration of the aqueous humor or lens.

The three sugars employed in these experiments are hexoses and have the same atomical formula. Dextrose and galactose are aldoses owing their reducing power to the aldehyde (COH) group, while lævulose is a ketose, in which the CO group is the reducing element.

Leaving aside for the moment the osmotic effect of the high concentration in which some of these sugars were employed, the differences noted in the toxic concentrations of these three sugars may be ascribed to the difference in their stereo-configuration, as shown by their structural formulæ.

DEXTROSE	LÆVULOSE	GALACTOSE
H—C=O	CH_2OH	H—C=O
H—C—OH	C=O	H—C—OH
HO—C—H	HO—C—H	HO—C—H
H—C—OH	H—C—OH	HO—C—H
H—C—OH	H—C—OH	H—C—OH
CH_2OH	CH_2OH	CH_2OH

It is known that lævulose is more readily utilized by the organism impaired in its carbohydrate metabolism, i.e., diabetes

mellitus. While it has been claimed that it is utilized less rapidly than dextrose by the living organism, it has also been claimed that it forms lactic acid more readily than glucose. There seems to be a biological difference in the use of these two sugars by the living organism. It has been shown that yeasts and moulds show greater growth in lævulose solution than in glucose solution of equal strength. On the other hand they consume dextrose preferentially from invert sugar. Lævulose is more readily utilized in tissue formation while dextrose is more readily used in respiratory processes and in fermentation. Postulating a greater ease of lactic acid formation and utilization for tissue growth for lævulose than for dextrose one may explain the anomalous behavior of the cultures with respect to these two sugars. As has been pointed out in the introductory remarks, cells in tissue culture grow under more primitive conditions than they do in the living body. It may well be that cells which revert to a more primitive metabolism are able to utilize lævulose more readily.

Galactose is much less readily utilized by the living organism than either dextrose or lævulose. It is also quite resistant to fermentation, only a few moulds and yeasts acting thus upon it. It is present in the cerebresides and in glucolipides of the white medulla of nerve fibres. It cannot be interconverted to dextrose *in vitro*.

The behavior of lens epithelium in tissue culture is in agreement with the chemical and biological properties of dextrose, lævulose and galactose.

Osmotic Tension

More difficult of explanation is the ease with which the tissue apparently adapts itself to the changed osmotic relationships. It is undetermined how much of the toxicity of the high concentrations of dextrose and læculose can be ascribed to the changed osmotic relationship, and how much to the actual toxicity of the sugar itself. Certainly if the changes in the osmotic concentration play a decided part then lævulose must be more readily utilized and assimilated in the higher concentration than dextrose. A concentration of 861 mgms. per 100 ccm. of medium proved toxic in the case of dextrose whereas a concentration of 1,660 mgms. per 100 ccm. of medium was reached when lævulose was used before a like toxic effect could be noticed. Expressing the

toxic concentrations of the three sugars in terms of millimoles, one obtains the following:

Lævulose	97.8 millimoles
Dextrose	47.8 millimoles
Galactose	29.5 millimoles

The usual concentration of dextrose in the culture medium is approximately 160 mgms. per 100 ccm. of 8.9 millimoles. If one assumes that the tissue readily equilibrates this difference in osmotic pressure without change in cell area, how is one to explain the absence of untoward reaction to greatly reduced dextrose concentration in the experimental medium? In the last of these series of experiments the dextrose concentration of the experimental medium was reduced to 12 mgms. per 100 ccm. or approximately 0.67 millimols, about 1/90 the concentration of the highest non-toxic lævulose concentration (Table 4).

Metabolism of Dextrose *"In Vitro"*

Tissue cultured *"in vitro"* derives its energy mainly from the metabolism of dextrose, part of which is broken down to carbon dioxide and water and part of which is glucolized to lactic acid. Krontowski (loc. cit.) showed that if dextrose is present in the medium in a concentration of 130 mgms. per 100 ccm. about 70 mgms. per 100 ccm. are used by the tissue in forty-eight hours and that about 40% of this amount is transformed to lactic acid. He also showed that in the first twenty-four hours there is greater sugar utilization. About two-thirds of the total amount are used in the first twenty-four-hour period. In the second twenty-four-hour period there is increased growth with decreased sugar utilization. The change in hydrogen ion concentration which the tissue culture undergoes is due primarily to the formation of lactic acid and the carbon dioxide set free by its action on the bicarbonate of the medium and only secondarily to the carbon dioxide resulting from the respiratory processes of the tissue culture. With a reduced dextrose concentration in the medium there is less lactic acid formation and hence less change in acidity.

The dilution of the embryonic extract and plasma necessary to obtain the extremes in concentrations of the sugars aimed at may also play a part. While the controls were subjected to a like reduction, the increased fragility of the growth most likely

rendered the tissue more sensitive to changes in environment.

If all these factors are taken into account the extraordinary adaptability of lens epithelium growing *in vitro* to changes in dextrose concentrations as well as to changes in kind of hexose in culture medium is evident.

The Effect of Acetone and Beta-Hydroxy-Butyric Acid

Table 5—*Average Concentrations of Ketone Bodies in Diabetes*

Severity of Diabetes	*Acetone* MGMS. PER 100CC. BLOOD	*Oxy-Butyric Acid* MGMS. PER 100CC.
Very Mild	1.5 to 3.5	4.5 to 9
Mild	4 to 6	12.5 to 18
Moderately Severe	7 to 10	20 to 30
Severe	10 to 20	30 to 60
Very Severe	20 to 40	60 to 120

Table 6

EXPERIMENT NO.	CONCENTRATIONS IN MGMS. PER 100 CCM. *Acetone*	*Beta-Hydroxy-Butyric Acid*	APPEARANCE OF EXPERIMENTAL AS COMPARED TO CONTROLS	*Remarks*
49	1.-	3.-	No difference.	
50	3.-	9.-	No difference.	
52	6.-	18.-	No difference.	
53	8.-	24.-	Controls better, larger area of growth, fewer vacuoles and degenerated cells.	
57	10.-	30.-	Controls better in growth and condition.	Experimental cultures recovered after transfer in normal medium.
60	15.-	45.-	Controls much better, experiments show increase in gran. vacuoles, diminished growth.	
62	20.-	60.-	Controls better.	This was a poor set. Controls also in rather poor condition.
64	30.-	90.-	Controls much better and difference between controls and experiments increases on transfer to same medium. Not actually toxic in three passages.	
68	40.-	120.-	Cultures died in 48 hours.	

Judging from these experiments the presence of these two ketones in concentrations comparable to a mild ketonæmia such as may exist in well controlled diabetes mellitus has no effect on the growth of lens epithelium *in vitro*. Beginning with a concentration of 8 mgms. acetone and 24 mgms. beta-hydroxy-butyric acid per 100 ccm. of medium a slight retardation in the growth

of the tissue may be noted, if not within the first forty-eight hours at least after the first transfer to the same medium. The growth-inhibiting effect increases with increasing concentrations. The presence of the two ketones in concentrations comparable to the ketonæmia of very severe uncontrolled diabetes mellitus, i.e., 40 mgms. acetone and 120 mgms. beta-hydroxy-butyric acid per 100 ccm. of medium killed the tissue within the first forty-eight-hour period. Figures 1 and 2 show the appearance of controls and tissues subjected to these higher concentrations.

The increasing growth-inhibiting effect of the ketones on lens epithelium cultures is shown in Table 7.

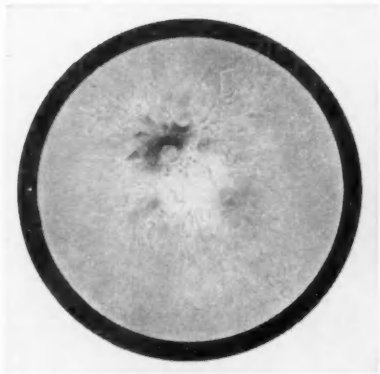


FIG. 1-A—Control Culture
Lens Epithelium
Control 64 × 3 — 2nd passage.
Obj. 4 Ocu. 5 × 1-21-32

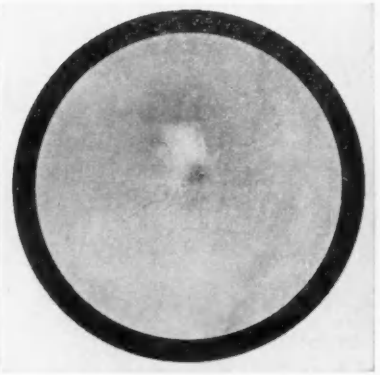


FIG. 1-B—Experimental Culture
Lens Epithelium
Experiment 64-4 — 2nd passage.
Obj. 4 Ocu. 5 × 1-21-32

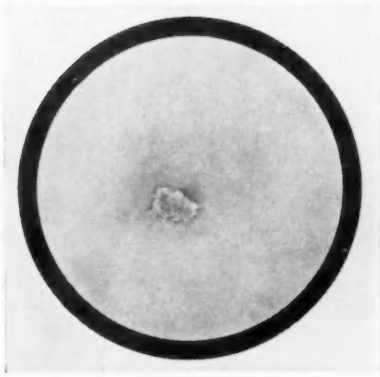


FIG. 2-A—Control Culture
Lens Epithelium
Control 68-7 — 48-hr. growth
Obj. 4 Ocu. 5 × 2-3-32

FIG. 2-B—Experimental Culture
Lens Epithelium
Experiment 68-6 — 48 hr. growth.
Obj. 4 Ocu. 5 × 2-3-32

TABLE 7

EXPERIMENT NO.	CONCENTRATION IN MGMS. PER 100 CCM. OF MEDIUM *Acetone*	*Beta-Hydroxy-Butyric Acid*	TOTAL NO. OF TRANSFERS	INJURY EFFECT NOTED AFTER TRANSFER NO.	DEATH OF TISSUE AFTER TRANSFER NO.	REMARKS
80	4.-	12.-	5	None	None	No difference between experiments and controls.
55	8.-	24.-	5	.....	.....	While the controls were slightly better after the first 48 hrs. this difference did not increase on subsequent transfers.
76	8.-	24.-	10	7th	10th	After the seventh transfer to same medium the difference between experiments and controls became increasingly noticeable, and after the 10th passage tissues were dead.
72	15.-	45.-	10	5th	10th	Experiments showed slight increase in granules at first. After the fifth passage to same medium difference in condition became more marked, and tissue was dead after the tenth passage (23 days of life).
78	20.-	60.-	6	3rd	6th	After the third transfer to same medium there was marked difference between controls and experimental cultures, and after the sixth passage (15 days) tissues were dead.
67	30.-	90.-	5	1st	5th	After the first 48 hrs., the experimental cultures showed smaller area of growth. This became more and more marked, granule formation as well, tissue dead after fifth transfer.

The cumulative action in the slight retardation of growth due to the presence of 8 mgms. acetone and 24 mgms. beta-hydroxy-butyric acid per 100 ccm. of medium manifested itself only after the seventh passage, but caused death after the tenth transfer. When the ketone concentration was increased to 15 mgms. acetone and 45 mgms. beta-hydroxy-butyric acid per 100 ccm., the growth-retarding action manifested itself after the fifth transfer and caused death after the tenth transfer. Twenty mgms. of acetone and 60 mgms. of beta-hydroxy-butyric acid per 100 ccm. of medium showed a noticeable difference in growth and condition after the third transfer and total inhibition and death after the sixth passage. These intervals were further reduced to a difference in growth and appearance after the first passage, and death after the fifth transfer, when the concentrations of the ketones were increased to 30 resp. 90 mgms. per 100 ccm. Figures 3 and 4 show two sets of these experiments.

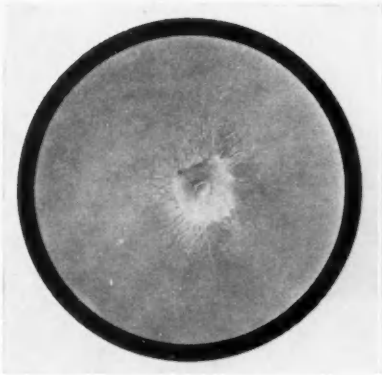


FIG. 3-A—Control Culture
Lens Epithelium
Control 72-3 — 7th passage.
Obj. 4 Ocu. 5 × 2-19-32

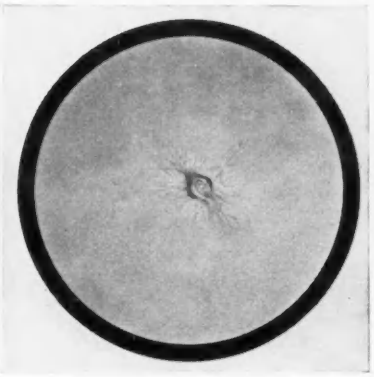


FIG. 3-B—Experimental Culture
Lens Epithelium
Experiment 72-4 — 7th passage.
Obj. 4 Ocu. 5 × 2-9-32

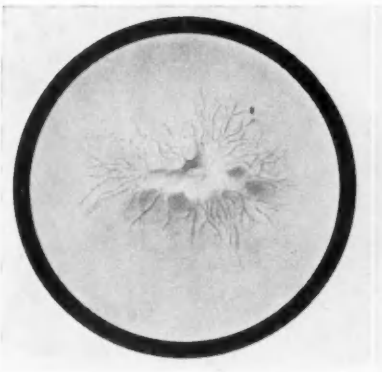


FIG. 4-A—Control Culture
Lens Epithelium
Control 78-5 — 3rd passage.
Obj. 4 Ocu. 5 × 2-24-32

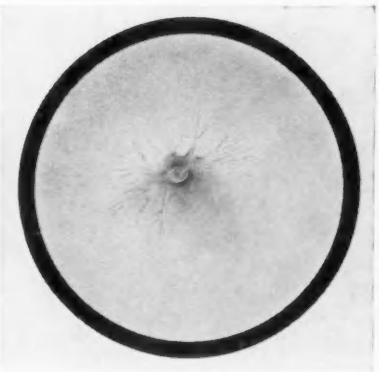
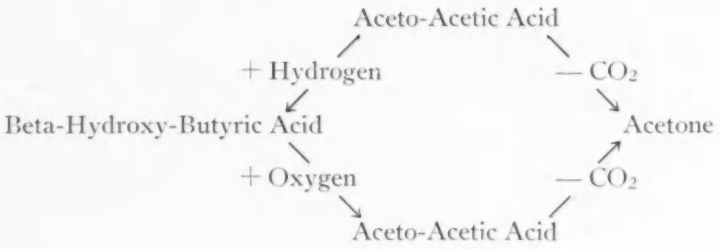


FIG. 4-B—Experimental Culture
Lens Epithelium
Experiment 78-6 — 3rd passage.
Obj. 4 Ocu. 5 × 2-24-32

Discussion of the Effect of Ketone Bodies on Lens Epithelium

Of the three ketone bodies which normally are present only in traces in blood and urine, acetone and aceto-acetic acid are volatile, while beta-hydroxy-butyric acid is a non-volatile substance. Aceto-acetic acid, which may be regarded as the parent substance is very unstable and is readily transformed to acetone by the loss of carbon dioxide. Aceto-acetic acid is reduced

concentrations of acetone and beta-hydroxy-butyric acid for the in the liver to beta-hydroxy-butyric acid, which, in turn, may be oxidized to the aceto-acetic acid. The cycle between these three bodies may, therefore, be represented as follows:



Normally the ketone bodies, acetone and beta-hydroxy-butyric acid may be present in minimal traces in the blood or reach a maximal concentration of 2 mgms. resp. 5.0 mgms. per 100 ccm. In infectious diseases, as well as in starvation their concentration in the blood may be increased to a maximum of 3.5 mgms. for acetone and 9.0 mgms. for the beta-hydroxy-butyric acid in 100 ccm. of blood. In certain liver conditions, such as yellow atrophy, carcinoma and cirrhosis, a like moderate ketonæmia may be present. In none of these conditions does one encounter the high values found in uncontrolled severe diabetes mellitus or the still higher values of diabetic coma. A mild ketonæmia is often associated with diabetes mellitus in its less-severe form.

The ketones are readily diffusible. Acetone on account of its volatility is excreted by the lungs as well as by the kidneys, while beta-hydroxy-butyric acid is excreted by the kidneys. The ketones pass through the capillary endothelium and are distributed in the tissue fluids, cells, and humors. Thus it has been shown that acetone appears in the spinal fluid of rabbits thirty minutes after its injection and that from an original concentration of about one-third of that in the blood, the spinal fluid acetone-content rises until it attains a higher concentration than in the blood.[34] It is reasonable to assume that the ketone bodies appear in the aqueous and vitreous humors. These experiments may, therefore, indicate that ketonæmia plays a part in the tendency of diabetes to cataract formation.

The lens cells were growing well when the experiments were made and, as the results show, were able to stand even the higher

first period of exposure, succumbing to the toxic influence only after the influence had continued for some time. The concentrations of very mild and mild types of diabetes has no apparent toxic effect on the cells, while those of the moderately severe had to be continued twenty-five days to produce the change. The concentrations of the severe stage produce toxic effect in fifteen days, while the very severe type concentrations bring about this effect in five days, and if the strength is increased to 40 mgms. acetone and 120 mgms. of beta-hydroxy-butyric acid, the cultures are killed and no growth results.

There evidently is a cumulative effect which is not manifested early in the weaker concentrations of these by-products of abnormal fat metabolism, which have a deleterious effect on the cells of the lens if the action is continued long enough, while the toxic effect is speeded up according to the severity of the disease, reaching a point in the most severe types in which it is impossible for the lens cells to continue life.

There is a possibility that, in the living state, all of the concentration of these materials does not reach the aqueous humor or that with active removal of by-products the effect would be less than with cells locked up *in vitro* circulation but with a change in nutrient medium every forty-eight hours.

All cases of severe and very severe diabetes do not develop cataracts. Some of these are in the severe stage for only a few days, being saved by heroic methods to combat the acidosis. Undoubtedly, if the severe stage were continued longer, more cases of rapid formation of cataracts would be seen. Fortunately, the handling of such cases is better understood and the severe stage is not continued long enough for the sensitive tissues, such as the lens, to suffer irreparable damage.

In many the effect of a long-continued low concentration of acetone and beta-hydroxy-butyric acid does undoubtedly cause changes in the lens epithelium or lens protein. These and other by-products of abnormal carbohydrate and fat metabolism may well be responsible for the lens changes of diabetes rather than simply the presence of increased dextrose content.

Summary and Conclusions

The effect of varying concentrations of dextrose, lævulose and galactose in the nutrient medium used for cultivation of lens

epithelium *"in vitro"* has been studied. Controlled experiments demonstrated that increases in the dextrose content produced no real difference in the health of the cells or in the growth factor until a concentration of 578 mgms. per 100 ccm. of medium was reached. Above this the dextrose was toxic. Lævulose reached the toxic point only at as high a figure as 1,660 mgms. Galactose proved toxic at 333 mgms. per 100 ccm.

The sugar content of the medium was reduced in another series of experiments to as low as 12 mgms. of dextrose per 100 ccm. Both the increased and decreased concentration experiments demonstrate the remarkable adaptability of lens epithelium *in vitro* to changes in sugar concentration and to the accompanying osmotic tension changes.

The effect of the two ketone bodies, acetone and beta-hydroxy-butyric acid, was studied. In the weaker concentrations the toxic action appeared only after repeated periods of exposure. The effect increases with concentrations until the toxic action becomes apparent within the first twenty-four hours. There is a possibility that long continued ketonæmia may be a factor in the tendency of diabetics to cataract.

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FIXATION AND VOLUNTARY NYSTAGMUS; A CLINICAL STUDY

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FIXATION nystagmus, by which we mean nystagmus resulting after fixing intently for some time on an object at rest, whether situated far or near, directly ahead or at an angle, is extremely rare. We have been unable to find reports of any such cases in either the English or American literature. However, a few such cases have been mentioned by the Germans and the subject is briefly discussed by Spiegel and Sommer,[1] Wilbrand-Saenger,[2] Brückner,[3] Oppenheim[4] and Fuchs.[5]

Voluntary nystagmus, on the contrary, while relatively rare, has been discussed for a long period of time, and to date there are over thirty cases which have found their way into the literature of the various countries. This condition is understood to be the production of a nystagmus by will power alone; that is, by concentrating on the idea and desiring the eyes to oscillate, when the motion results. This, to be of the true type, is independent of fixing the gaze in any direction or upon any object.

It has been our good fortune during the past year to observe two cases of nystagmus, one conforming in every respect to the fixation type as defined above, and the other to the pure form of what is defined in the literature as "voluntary nystagmus."

REPORT OF CASES

Case 1—J. C. S., a male, aged 13, attends school; he is an orphan.

Family History: This boy has been an inmate of an orphanage since babyhood. He has never seen either parent and nothing is known of them or of his heredity. He has always been well physically and mentally and has taken an active interest in sports at school. There is no history of encephalitis and no symptom of an attack.

Physical Examination: His physical development has been normal. His pupils are round, equal and react promptly to light and in accommodation. The eye movements, media and eye grounds are normal. The visual fields for form and color are normal in both eyes. The vision is 20/20 in each eye; there is no refractive error present and the muscle balance is normal. The special senses are normal. Response to caloric test is normal in both ears. Stigmata of degeneration are absent. The tendon reflexes of both the upper and lower extremities are present, equal and normal in response. Sensory disturbances are absent. Station and gait are normal. There are no evidences of any organic nervous diseases. His blood pressure is 80 mm. Hg. systolic, 60 mm. Hg. diastolic. His heart, lungs and abdomen are normal.

The laboratory examination reveals a normal urine, normal red and white blood counts, normal hemoglobin, a negative blood Wassermann reaction, and normal blood chemistry.

Mentally this boy has no vicious habits; he has a pleasing personality; he sleeps well, he is active but is mentally retarded, due in part to the oscillations of his eyes during the past three years when attempting to read.

About three years ago he observed that while studying, occasionally his eyes, after fixing on an object, would suddenly "shake." This would be followed shortly by a painful closure of his eyes, and the motion would stop. Should he repeat his effort at reading, after a short time this phenomenon would be repeated. He has tried to stop this in school, but it has increased so that he has been changed from close work to the manual training department, where his work does not require intense or long fixation upon objects. This oscillation of the eyes can be brought on by fixing on an object in any direction, far or near. The oscillations are of a horizontal and rotary-horizontal mixture; they are rhythmical; the amplitude does not exceed 1 mm. and the frequency is between four and five hundred hundred per minute. The duration is but a few seconds, and the end is preceded by a rapid convergence of the eyes, associated with a painful closure of the lids and a shaking of the head. The eyes are then normal until he fixes on another object, when the process recurs. These oscillations can be produced not only when fixing upon far or near objects with both eyes open, but when either eye is covered; also after a mydriatic, or prisms with

bases set in any direction. The element of wish does not at any time play a factor in the production of the oscillations, and he cannot stop them except by convergence and closure of the eyes, both of which are involuntary.

Case 2—D. P., a female, aged 21; single, a trained nurse.

History: She observed, as a small child, when trying to develop some trick for the amusement of her playmates, that she could make her eyes "dance" at will. She recalls distinctly the onset and states that she practiced doing this so that she could use it in parlor entertainment. To produce this condition, all that is required is to concentrate on the idea of making her eyes "jump," and shortly a nystagmus of rotary type, with some elements of a horizontal nature, will result. This nystagmus or oscillation can be started or stopped at will and can be produced when one or both eyes are closed, when both eyes move as though they were open. This oscillation can be produced when looking straight ahead or when looking to either side, but it is easier when looking toward the left than to the right. Prisms placed before the eyes with bases in any position have no effect upon the oscillations. These oscillations are rhythmical, of short duration, and occur about three hundred times a minute. Should she "will" to continue the oscillations over many seconds, her eyes feel tired. However, after a few moments' rest, she can reproduce the condition.

The vision in each eye is 20/20. The muscle balance is normal. The media and fundi are normal. Fields for vision and color are normal. The pupils are round, equal and react. There is a slight hyperopia of +0.37 sphere present in each eye.

We find her family history negative for any nervous or mental disease. She has five sisters and one brother, alive and well. Her father died from a post-operative complication. The mother is alive and well. She had the usual children's diseases, and three years ago her appendix was removed. With the onset of maturity, she began having severe headaches of a migraine type. These occur two or three times per month and last from two to four days; they begin usually on the left side of the head, and terminate with severe nausea and vomiting. They are not associated with her menses and have no influence upon her eye movements. There is no history of encephalitis, and no symptoms of a previous attack are found.

Physical examination reveals a well-developed young woman, whose weight is 110 pounds. Blood pressure is 110 mm. Hg. systolic, 70 mm. Hg. diastolic. The heart and lungs are normal. The special senses are normal. Her tendon reflexes are all present and equal. There are no abnormal toe signs. There is no sensory disturbance of any type. There is no tremor of the fingers. Her station and gait are normal. Caloric tests are normal. Laboratory examination reveals nothing which deviates from the normal. Her blood Wassermann reaction is negative.

Mentally, she is sociable, a good mixer, is diligent; she has a pleasing personality and has no tendency to any eccentricities.

Discussion

It will be observed that the two cases differ in their mode of inducing nystagmus. The course is different and the episode terminates differently. In one case nystagmus induced by fixation comes on independently of any wish, but deep concentration apparently slightly hastens the onset. In the other case, fixation is not necessary to produce the motion; in fact, she can induce this phenomenon voluntarily by closing both eyes, or one eye, or by looking in any direction. The desire to have the eyes "dance" is sufficient, if she concentrates but a moment. Her case differs somewhat from many of the published cases in that respect. Several of those in the literature are what we should regard as a mixture of voluntary and fixation nystagmus. Most of the cases, however, come under the voluntary type. Dodd's[6] case must fix on an object, then wish for a moment, before the oscillations begin. Friedenwald's[7] case began as an ocular nystagmus of the optokinetic type, and later he found that he could produce some motion of the eyes voluntarily by looking in any direction. Waddy's[8] case was obliged to stare fixedly into the distance, and then by deep concentration and desire he could produce oscillations of the left eye alone. These were the only cases in which the element of any combination of fixation and a voluntary act were combined.

More or less pure cases of voluntary nystagmus have been described by Williams,[9] Lafon and Bonnet,[10] Coppez,[11] Pyle,[12] Benson,[13] Raehlmann,[14] Sterling,[15] Major Elliott,[16] Smith,[17] Lawson,[18] Gamble,[19] Brückner,[3] Wilbrand-Saenger[2] and others.

In any effort to explain these types of nystagmus one must of necessity consider the explanation of nystagmus in general. Many theories are postulated, but as yet no entirely satisfactory explanation of nystagmus is at hand. By nystagmus we understand a disturbance of the static condition of the eyes wherein the normal associated movements of the globes become involuntarily rhythmic and present an associated tremor. Nystagmus in general is believed to orginate from three sources: (a) Ocular; (b) Vestibular; (c) Central (cerebellar or cerebral). Malcangi[20] and Posey and Spiller.[21]

In this communication we are especially concerned with the so-called ocular type, because we feel that our cases come under what various authors have agreed upon as belonging to some disturbance of optic origin or of the neuromuscular mechanism of the eye. However, the various theories of nystagmus will first be discussed.

The remarkable regularity and parallelism in the movements in nystagmus show that we have to deal with a perversion of the centers for parallel and parallel-rotary movements, and not with peripheral lesions of the muscles themselves or their nerves. (Posey and Spiller[21]).

It is possible that nystagmus may be produced by alternating, just as normal fixation is produced by simultaneous discharges of motor energy from the two sides of the brain. These centers are located in the cortex, the exact area is not yet isolated, and when impulses from both the right and the left centers follow each other in rapid succession, nystagmus results. Nystagmus may be interpreted as a type of imperfect or perverted fixation. In line with this theory unilateral or disassociated types of nystagmus are those in which the reflex discharge from the associated centers which cause the oscillations are in some way allowed to affect one eye more than the other. Occasionally a muscular anomaly will explain the inequality of response. Fuchs[5] and Duane[22] express the principles of the above in their writings.

Malcangi believes that whatever may be the nature of nystagmus, it is necessary to presuppose the existence of a rhythmic center which coordinates ocular movements, the changes of which result in the development of nystagmus. The stimuli which affect this rhythmic center and which activate it are those which control the motor apparatus of the globe. These arise from tonic

association centers that regulate the associated movements of the eyes and preside over the function of binocular vision and of fusion of images in all directions of view. These associated centers receive their impressions in turn by various pathways, (the vestibule, the auditive and the ocular) and may give rise to forms of vestibular, auditory or optical nystagmus. These impressions also are in relation with the cerebral cortex, which presides over the voluntary movements of the eyes. In these centers all the various stimuli are coordinated and subordinated.

Ohm[23] feels that there is not a great deal known about the cause of nystagmus. He feels that in mountain dweller's nystagmus and in voluntary nystagmus there are three possible causes, all interwoven, — optical, vestibular and cerebral. Whether these arise from primary centers or from the cortex, he is not sure. He also demonstrated that concentration influences the character of the nystagmus in either case, showing the mental influence from the frontal lobe. He found that if the patient looks fixedly at an object situated at an angle, right or left, 10-cm. distant, that there was an increase in the frequency of the nystagmus and a decrease in its amplitude.

Spiegel and Sommer[1] believe that fixation nystagmus may occur in normal people and also in people with degenerative stigmata. They say it is best produced when looking at an angle, for then there is an imbalance in the eye muscles in which the agonist has a stronger contraction than the antagonist, which results in this type of person in an oscillation, or a nystagmus of the eyes of varying severity, and it may occur in several directions, bilateral, or a combination of these with rotary. They further believe that there is a similarity between muscle tremor of the skeletal muscles and the eye oscillations. If a person with normal muscle eye balance fixes intently for one or two minutes, the eyes usually oscillate; or in fatigue of the body muscles, as by holding fast to an object for some minutes, there results a muscle tremor. In the eyes it is called nystagmus. In both instances repetition results in the tremor being more quickly produced and the amplitude is greater and the rhythm faster. All these can be easily produced with repetition in neurotic people. It has also been brought out in certain toxic states, that the more energetic and the deeper the concentration on the process, the more frequently and easily the condition is produced.

They, therefore, regard the phenomenon purely on a functional basis with fatigue as the precipitating factor. They feel that voluntary nystagmus can be produced by normal people, but not often; usually in individuals with some stigmata. They regard it as a type of ocular nystagmus.

Lafon[24] regards voluntary nystagmus as a disturbance of the tonic equilibrium between the controlled muscles, due to an excessive convergence. He feels that all voluntary movements of the eyes are associated ones, governed by the function of direction and convergence. These functions are of cortical origin, arising not from one center, but from several associated sensorial centers scattered through the cortex. They are coordinated by centers located probably in the mesocephalon, which transmits them to the oculo-motor nucleus. There may exist, also, supranuclear, intermediary and subcortical centers.

Brückner,[3] after reviewing all the cases of voluntary nystagmus up to 1917, and adding several of his own, agrees with our findings that not all cases were purely voluntary. He is not certain that the term "voluntary" explains the response. It is his opinion that there is a strong inervation impulse sent to the muscles of both eyes, after which the eyes begin to oscillate. He does not think it fully represents a voluntary act or response. He believes that only a few people can produce the phenomenon, and that such individuals are susceptible to strong innervation and strong impulses affecting the eyes.

It would appear, then, that there are really two schools of thought relative to the mechanism of these types of nystagmus; one that they result wholly from physiologic responses from stimuli affecting the muscles of the eyes of neurotic or hysterical types, or in those held susceptible to strong impulses directed to the eyes with the idea of oscillation or motion in mind. Others think it a fatigue process, while still others liken it to a tremor process of the skeletal muscles in fatigue.

The second school feels that it is a part and parcel of stimuli affecting the cerebral coordinating and rhythmic centers for the eyes, which are not definitely fixed centers, but are located in the cortex and subcortical centers in the mesocephalon.

Comment

It is evident to us that both our cases belong to what is regarded as the ocular type of nystagmus, though they present somewhat different manifestations of this rather unique condition. Ocular nystagmus manifests itself in many forms, which grouped together may be regarded as the result of some change in the eye itself; in some disturbance of its neuromuscular mechanism; or in a faulty position of the eye, in very faulty refraction, or from some degenerative defect of the eye. The causative factors thus may be either physiologic or pathologic.

The nystagmus resulting from position (endstellungs nystagmus) such as fixing the eyes in the limit of the field of vision; the optokinetic nystagmus, due to observing moving objects, as from the car window of a rapidly moving train, or observing high waterfalls, are physiologic; while those types dependent upon imperfect light, plus bad position of the eyes, as in miner's nystagmus, mountain dweller's nystagmus, spasmus nutans, and those due to impaired vision of congenital origin, or to disorders of the acuity of vision or of vision itself, as in amblyopia, — are all regarded as pathologic.

Voluntary and fixation nystagmus, latent unilateral and dissociated nystagmus are classed along with the ocular types, and have a more or less common factor in their production, but still the mechanism of the phenomenon in the various types is somewhat different.

In our voluntary case, the nystagmus appears to be the result of a sudden strong impulse applied to the muscles of both eyes with the idea of oscillation in mind, and that by the frequent repetition of these stimuli there has resulted the easy production of the movements at will. All this, however, presupposes a rhythmic center in the brain to coordinate the ocular movements. The stimuli in the form of wish and concentration, affect this rhythmic center, in all probability in such a way that a change in the equilibrium is made and we get alternating rather than simultaneous discharges of motor energy from the two sides of the brain, and the nystagmus results. It is barely possible, however, that fatigue may play a part in this case, as she usually turns the eyes somewhat to one side before the onset; in this way the imbalance of the agonist and antagonist muscles, due to fatigue, may permit the oscillations or tremor

of the ocular muscles to ensue, with the resultant nystagmus. This belief is strengthened when we bear in mind the completely negative findings in this case in the way of refractive errors, muscle balance, field changes, and the absence of any demonstrable organic eye or brain lesion.

In our fixation case the mechanism is somewhat different, as here there is no wish element, but merely the fact of fixation on an object at any point in the visual field. As previously it took longer for our patient to induce the nystagmus than at present, it would thus suggest that there was a strong element of fatigue in the case, wherein the muscles for maintaining the static condition of the globe become unequally fatigued and an oscillation results. This rapidly increase until convergence soon occurs, which becomes painful; this is followed by a sudden closure of the lids, the oscillations suddenly cease, and the eyes immediately resume their normal position. In this way it can be analogous to the muscle tremor theory of Spiegel and Sommer, and the affair be more the result of a neuromuscular mechanism than the result of a strong mental stimulus affecting the rhythmic center of the cortex in such a way as to give rapid alternating, rather than simultaneous discharges from this center. Again in this case we have the complete absence of all organic ocular and neurologic signs, a perfectly normal muscle balance of the eyes, with no refractive errors and no disturbance of vision whatever.

The final analysis, would indicate that both of these cases are functional, rather than organic in origin, but that they are produced by somewhat different mechanisms; one the result of a strong desire, with concentration; the other by fixation on an object, before the oscillations begin. The one terminates at will, while the other only after rapid convergence and closure of the lids. The duration of each is from twenty to thirty seconds, the amplitude and rapidity of the oscillations are about equal in each, and both feel fatigue about the eyes after the phenomenon has passed.

Summary

1. Two cases of nystagmus are herein described; one, the so-called fixation type, and the other the voluntary type.
2. The fixation case produces nystagmus by fixing on an object in any point of the visual field, when soon a rapid oscillation

occurs, which is followed by sharp convergence and pain; then a sudden closure of the lids, with immediate return of the eyes to normal.

3. The voluntary case is that of a young woman, who, by concentrating on the wish to oscillate her eyes, or to make them "dance," with or without the eyes closed, or when looking in any direction, there results an oscillation of both eyes. This can be stopped at will, but if unchecked the eyes fatigue in about thirty seconds, when the process stops.
4. We feel that there is probably the same underlying principle in the production of both types of nystagmus. The sending of strong stimuli from the rhythmical center of the cortex, producing alternating rather than simultaneous discharges of energy, is one explanation.
5. The process may also be explained as the result of fatigue of the muscles resulting from the primary fixation or gaze, plus frequent repetition of the process, with the resulting imbalance between the agonist and antagonist muscles when oscillations follow.
6. In either event, we feel that both types belong to what is known as ocular nystagmus, and that both are functional in origin.

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DISCUSSION

Dr. John H. Dunnington, New York: Fixation nystagmus must not be as rare as the paucity of the literature would make one think. Its presence has been known for many years, but still no very satisfactory explanation has been offered. Duane considered nystagmus to be due to a perversion of the centers for parallel and parallel rotatory movements and referred to it as a kind of perverted and imperfect fixation. If nystagmus is due to a perversion of fixation, it is reasonable to expect that in some cases the oscillations will become worse on attempting fixation. It is generally understood that in nystagmus the oscillations become least in that position of the eyes which for this case involves the least strain upon the eye muscles, or they are least under that condition of vision which implies the least confusion of sight. Therefore, it is proper to assume that in fixation nystagmus the attempt to fixate either confuses the vision or throws the greatest strain upon the eye muscles. Macular lesions, central corneal or lenticular opacities are frequently encountered in this type of nystagmus. All of these conditions render fixation more difficult. Although Doctor Luhr's case shows no such pathological condition it seems reasonable to assume that the vision becomes most confused on attempting fixation and that this in turn produces the nystagmus.

Voluntary nystagmus is unique and its occurrence has been frequently noted in the literature. Bruckner, an oculist who possessed this power, has described the sensations he experienced during the oscillations, as those of marked innervation of convergence and of accommodation and compared them to those felt when the arm is held in strong flexion. Bruckner, Weekers and others have referred to the contraction of the pupil during the oscillations. These observations would lead one to think that the nystagmus resulted from the impairment of vision produced by the transitory spasm of accommodation yet Smith has recorded a case of voluntary nystagmus in which the oscillations could be produced while the eyes were closed. Furthermore, the unilateral cases reported by Pyle and Ball are not easily explained by the visual theory. With such conflicting evidence, it seems most plausible to assume that Doctor Luhr is

correct in his contention that there is a center controlling the rhythmic movements of the eyes, and that voluntary nystagmus results from some functional disturbance of this center.

Dr. E. E. Blaauw, Buffalo, New York: I have had the privilege of seeing the case of Dr. Luhr a couple of times and I must say that the reality makes a different impression from the picture. The thing that strikes me is, is this plain nystagmus, or rather pseudonystagmus belonging to another category of muscular phenomena? In the picture the action of surrounding muscles, as for instance, the orbicularis bulbi and the frontal muscles, shows up quite distinctly and prominently.

Dr. Luhr mentions the name of Ohm. He has devoted much of his energy in the last twenty years in this direction. The old idea of Arlt that because the macular region was involved the patient wanted to shift his eyes to see better, is strongly contradicted by Ohm, who directs attention to the cases of monocular nystagmus which shows monolaterally — when the eye is in motion — a distinct diminution of vision, which improves as soon as the eyes are used together and thus lose the nystagmus.

Dr. Edward Jackson, Denver: I had the opportunity of seeing these cases when Dr. Luhr showed them on Monday in Buffalo. With the review that has been given of the previous literature they become more interesting. I was impressed when the two cases were properly brought together, that there is an element of voluntary innervation that helps produce nystagmus in both cases. In the fixation nystagmus, the voluntary effort is that of fixation; and in the one which might be called involuntary nystagmus, a certain psychological element enters; and the innervation has probably been acquired by practice.

The whole incident reminds me of some experiments that were made many years ago, when I was a recent graduate of the University of Pennsylvania. Two young men, Dr. A. J. Parker, who was prosector for the professor of anatomy, and Dr. F. X. Dercum, who was subsequently Professor of Nervous Diseases in Jefferson Medical College, began experiments on themselves and then induced others to join them. They were brought to the notice of Dr. S. Weir Mitchell, who was much interested in them, but gave warning of a danger of continuing them, and they were discontinued on that account. The experiment has sufficient bearing on this matter of nystagmus to here speak of it.

When we were trying it we would sit around a table, four or six at the table — the experiment was done in the hall of the College of Physicians. We sat easily, each in his chair, holding our hands outstretched, the fingers just so they did not touch the table; each held them so as long as he could keep it up, making a decided effort to hold them still. Presently it became harder, the hands began to move, and to hold them still was no longer possible. That was a sort of nystagmus of the hands. Those who had practiced that exercise longest, and who had enough control to continue holding the hands still longest, were most affected when movement became involuntary. The motion was that of a trembling of the whole hand and upper extremities. In the case of Dr.

Parker and Dr. Dercum the jerking became so violent and so excessive that they would fall backwards on the floor. The danger Dr. Mitchell had in mind was the possibility of acquiring an epilepsy in that way. What he said to the young men who had started it influenced them enough so that they quit making this experiment. But it was repeated by a dozen medical students and young doctors, and the order of the phenomena was always the same. The hands were held still as long as possible, and when the quivering became involuntary it increased until the spell went off.

It seems to me that here is an experiment that ought to be connected with nystagmus. I have felt that poor vision was pretty closely connected with nystagmus, not merely as an effect. I have seen nystagmus in cases where there was not poor vision yet with an error of refraction, and a few cases in which the nystagmus gradually cleared up after correction of the error of refraction; and some in which the nystagmus decidedly diminished and remained so several years.

Some one remarked that nystagmus was more frequent than was suspected. Examining eyes for nystagmus with the ophthalmoscope, fixing on the back of the eye and trying to keep the eye fixed, will in a fair number of cases, cause the patient to develop a slight nystagmus; in other words with the ophthalmoscope it is possible to detect nystagmus that is very hard to find by any other method.

Dr. Alfred F. Luhr, closing: I wish to thank Dr. Dunnington, Dr. Blaauw and Dr. Jackson for their discussion of this paper. I was very much pleased to have had the opportunity of showing these two cases to Dr. Jackson while he was in Buffalo.

I do not know that I can say anything further so far as the etiology of these cases is concerned. In going over the literature we have attempted to apply to our cases the various theories that have been advanced for the mechanism of nystagmus.

ASTIGMATIC CHARTS VERSUS CROSSED CYLINDERS

Results in One Hundred Office Cases

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NO COMPARISON of two such tests is fair or valid without a careful statement of the technique employed. No matter how good the test when skilfully used, when used with a wrong technique it may be of little value or even positively misleading.

In all cases the taking of the history, the external examination, and the ophthalmoscopic examination precede the use of these tests. In most cases the astigmatic charts were used before the crossed cylinders.

TECHNIQUE WITH CHARTS

While the room is still darkened following the ophthalmoscopic examination, the patient is told to keep the eyes closed, and the trial frame is put on. Insert spherical lenses strong enough plus or weak enough minus to overcorrect the refractive errors about half a diopter more than the strongest meridian. For instance, if you guess that the refraction is about $+1.00 \bigcirc +.75$ cylinder Axis 90 you will insert a $+2.25$ sphere. But if you know for any reason (previous examination by yourself or someone else) that the above is very close to the true correction, $+1.75$ or $+2.00$ may be used to start with. If you use a spherical correction which is too weak, you invite the patient to use accommodation; and this he will be sure to do even if he has had cycloplegics instilled. One has a fair idea of the vision from the history and from taking the vision before the ophthalmoscopic examination was made.* These initial lenses will reduce

* Do not take the vision immediately before beginning to make the test, because that requires the patient to exert his accommodation. The accommodation is made to relax by being in a darkened room, especially with the eyes shut.

the vision about two lines on the chart,† hence they are called fogging lenses. For the average case fog to 6/10.

Place an opaque disc in the trial frame before one eye and begin the test for astigmatism by asking the patient to look at the chart with the many radiating lines.§

Ask the patient if the radiating lines look all alike, or if some are darker than the others, more distinct, stand out better. If he sees no difference, this may be because there *is* no astigmatism sufficient to make a difference, or it may be because he is not a good enough observer to detect the difference. To settle this question, place a minus cylinder before the fogging lens, say a — 50 cylinder, holding it now at 90°, now at 180°, or other angle. This, when placed in one position, will make the lines parallel to that position better focused, less burred, darker, more distinct. The patient should recognize this artificially produced astigmatism unless it happens that you have so placed the cylinder as to correct to some extent an astigmatism which he has but did not observe. When you reverse the axis, this should make the lines in the new direction more distinct. If the cylinder corrected an astigmatism when in the first position, it will increase this astigmatic error when reversed; so that if the patient fails to detect any difference in the lines in either position, he is a poor observer. Try a stronger cylinder, say a — 1., holding it at different angles and telling him which lines *should* look more distinct.

If the patient is under cycloplegia, or for any reason has no accommodation, you must be particularly careful about the strength of the fogging sphere. For example, if you have in the frame a + 2., and his true correction is + 1.75, if you hold

† It is desirable to use the charts designed by Ewing. The change in size from any one line to the next is the same throughout; it equals $\sqrt[5]{2}$ as recommended by John Green, Sr.

§ This chart should comprise a complete circle of 360° with lines 10° apart. The width of the lines should be equal to that of the letters of the 6/16 line. There should be the maximum possible contrast between the lines and their background; i. e., the lines the blackest black, the chart the whitest white. Those made by Bausch and Lomb are the most generally satisfactory of any that I have seen. They are made according to my design except that the white paint used is not as white (does not reflect as much light) as the paint I advised. The modification designed by Friedenwald is even more sensitive than mine when used on patients who have small errors of refraction and are good observers. On the rank and file of patients mine give better results. On good observers either chart will give satisfactory results.

a —.75 cylinder before this, it will make the lines parallel to its axis *less* distinct instead of more distinct, because it will make the power in that axis +1.25, while the opposite axis remains +2. +1.25 is farther from the true correction than +2. is.‡

It may take several minutes to teach the patient what he is to look for, and to train him in recognizing the changes which you produce by changing or reversing the cylinder. It will soon appear whether the case is one unsuited for the test. If because of poor vision, or poor power of observation, he fails to detect a —1., or if he is a patient with average visual acuity, a —.50 cylinder, do not waste time with this test.

When the patient says that he sees some of the lines more distinctly, go to the chart and help him to locate them exactly. If there is a group of these lines, then the center line is the true axis. If there are four lines, the true axis is between the two center lines. If the astigmatism is small in amount, the number of lines is larger and it is harder for the patient to decide where the center of the group is located. Some patients find it easier to say which lines are most blurred rather than which are most clear. Keep a watchful eye on the patient's head, because when he tilts it he tilts the apparent axis of his astigmatism.

In cases of irregular astigmatism there may be an irregular distribution of the clearer lines; e.g., 20° and 30° may seem fairly clear, 40° blurry, 50° clear. In regular astigmatism there is always a group fading away equally in both directions from the most clearly focused center.¶

It is not uncommon for the axis which is chosen with one fogging lens to be different with a lens of another power. Which

‡ One who has not had much experience with this test should try many experiments on himself, and on patients who are good observers, using spheres of different strength and noticing how they affect the appearance of the chart, and using cylinders of different strength and at different axes. When the answers are different from what you expected, it is especially important to find out why. Usually it is the accommodation which is playing tricks; often it is irregular astigmatism. Do not fall into the habit of explaining such answers as always due to the stupidity of the patient, since this is conclusive evidence of the stupidity of the examiner.

¶ It is one of the peculiar merits of this method of measuring errors of refraction that it enables one to determine, in cases where there is irregular astigmatism, the largest amount of regular astigmatism which can be corrected by our lenses. Of necessity, our lenses can correct only regular errors. It is the maximum regular component which we wish to determine, thus leaving the minimum of uncorrectible residue.

shall we choose? My custom is to complete the second part of the test, measuring the amount of astigmatism, and then return to the determination of the axis, using for the fogging lens a lens of the exact power of the stronger axis. For example, with + 2.50 the axis may be 120°, as nearly as can be determined. Having found that the amount of the astigmatism is + .50 cylinder, and that with + 1.50 sphere ⊃ + .50 cylinder the vision is 6/6, but with + 2.00 sphere ⊃ + .50 cylinder it is 6/10, the proper lens for fogging is 1.50 + .50 = 2. With this the axis may be 110°. If so it is probably nearer right than the axis 120° found with over-fogging. With + 1.75 the axis might be 105°. It is in cases like this that the use of the crossed cylinders is apt to be most helpful, if the answers with them are satisfactory.

This completes the first part of the test with the charts; namely, finding the axis of astigmatism. The cross on the second chart is now rotated so that one line corresponds with this axis. The patient is asked which line is more distinct and black, the one with the red end or the one with the black end.*

Minus cylinders are placed before the fogging lens with the axis parallel to the less distinct line of the cross. Since the sphere is a fogging lens, reducing its strength by minus cylinders will reduce the fog and make that line clearer. The cylinder which makes that line equal to the other line measures the amount of astigmatism. If the cylinder is a weak one, it will make the test easier and more accurate to use less fogging. If the cylinder is strong, it may be necessary to use more fogging. The need of this is determined very quickly by asking the patient to read the smallest letters he can. Holding a — .25 sphere before the combination should not only make the letters clearer but enable him to *read more*. Holding a + .25 sphere before should make him read less. When this is the case, the fogging is just about right for the final decision. This is made by holding a — .25 cylinder parallel to one axis and then to the other. The patient should promptly say that the line on the cross is more distinct which is parallel to this cylinder. Sometimes it is better to use a — .50 cylinder until the patient sees the point,

* A red disc on the end of one line and a black disc on the other make it easier for the patient to tell you which line is clearer; but be sure he does not think you wish to know which *disc* is clearer, but only which *line*.

since he may be afraid he is contradicting himself when you reverse the cylinder. Then use a — .25 and finally a — .12 cylinder. Most patients tell which way you hold the — .12 cylinder. This means that you have the astigmatism corrected, so that a cylinder .12 D stronger or .12 D weaker is too strong or too weak; that is, your correction is right to less than an eighth of a diopter. Sometimes a + .25 and + .12 cylinder are better than the minus cylinders for this final test, depending on whether the fogging is just right or insufficient.

Next, measure the astigmatism of the other eye. The patient, having had instruction and practice, answers more promptly and accurately.

Finally, measure the spherical correction. This should be done with both eyes at once. Place in the frame the cylinders found to correct the astigmatism, using a plus cylinder when the error is hypermytropic, and a minus cylinder only when it is myopic. Start with spheres which will give vision about 6/8. See if one eye is about as bad as the other with these lenses. Reduce the sphere until maximum vision is obtained. Holding + .50 over both eyes should reduce the vision at least one line. Holding it over one eye at a time should reduce that eye two lines. For example:

O.D. + 1.25 ⊃ + 0.75 Axis 85 V = 6/5+
O.S. + 1.00 ⊃ + 1.00 Axis 95 V = 6/5+
O.U. 6/4, +50 6/5 6/6 or 6/8 6/6 or 6/8

Where the pupils are small the + .50 reduces the V less than where the pupils are large.

Technique Followed in Using the Crossed Cylinders

The crossed cylinders should be mounted in the approved way, with a round handle about five or six centimeters long, attached midway between the two axes, that is, 45° from each. This is essential for quick and accurate reversal of the axes of the crossed cylinder as it is held before the eye. The most frequently useful in my experience is the + .25 ⊃ — .25, next the + .50 ⊃ — .50; these total cylindric powers are .50 cylinder, and 1. cylinder, respectively.

The two steps in the use of the crossed cylinders are: first, determining the strength of the cylinder needed to correct astig-

matic error; second, determining the axis. In this series of observations, since the astigmatism had usually been measured first by the charts, the approximate axis was known. A combination correcting approximately the errors is placed before the eye, the other eye being blanked. The spherical component should be strong enough plus to cause a little fogging; for example, if the corrected vision is 6/4, fog to 6/5 or 6/6; if it is 6/6, fog to 6/8, and so forth.

The first test is to determine whether the approximate axis can be improved. If the vision is good and the cylinder is not large, I prefer the $+.25 \bigcirc -.25$. If the vision is rather poor, or if the cylinder is strong, use the $+.50 \bigcirc -.50$. The crossed cylinder is held with *the handle in line with the axis of the cylinder* in the trial frame being tested. The algebraic sum of the crossed cylinder in this position plus the correction in the trial frame is a new combination with the axis shifted. When the crossed cylinder is reversed, the axis is shifted in the other direction. The patient is asked to read the letters and tell whether he can read smaller letters with the crossed cylinder in one position or in the other. If he sees no difference and proves it by reading the same each way, then the axis is correct by this test. If he sees better with the crossed cylinder in one position than in the other, move the cylinder in the trial frame a little toward that position. If it is a plus cylinder, move it toward the axis of the plus component of the crossed cylinder. If these axes are marked, the plus with a white dot, the minus with a red dot, it is easier to recognize them. It is important to realize the fact that the crossed cylinders reduce the clearness in *both* positions. The question is not: Does this glass make the letters clearer than without it? The question is: Are the letters clearer with the crossed cylinder in one position than in the other?

If one has not done so, he should try this on his own eyes. He will be surprised to see, when the correcting lens in the trial frame is not on the true axis, how well-marked a difference there is between the two positions of the crossed cylinder.

The second part of the test is to determine the strength of the cylinder needed to correct the astigmatism. Start with what you guess is an approximation to the correct power. The patient should be very slightly fogged. Hold the crossed cylinder with one axis coinciding with the axis of the cylinder in the trial

frame; see how far he can read; then very quickly reverse the crossed cylinder and see whether the letters are clearer or the reverse. If they are clearer when the axis of the plus component of the crossed cylinder coincides with the axis of the cylinder in the trial frame, this means that the cylinder in the trial frame should be made stronger plus, or weaker minus. Make the change and repeat the test. When the patient reports that he sees no difference in the letters, whether the crossed cylinder is held with the plus component or the minus component over the axis in the trial frame, then the power is correct by this test. Do not make the mistake of comparing the vision *with* crossed cylinder and *without*. Of course, it is better without. It is the two positions of the crossed cylinder that are to be compared. One increases the power of the cylinder in the trial frame by a certain amount, the other decreases it by the same amount. According to this method, it is assumed that increasing or decreasing the power of the true correcting cylinder by the same amount produces the same amount of blur.

Both the test by use of astigmatic charts and the test by crossed cylinders depend on the patient's power of observation. In the case of the charts the patient looks at the objects he has to compare and sees them simultaneously, or rather they are both before him and he can look from one to the other as often as he wishes and as long as he wishes. In the case of the crossed cylinder test, the objects are exposed successively, first one and then the other. The two are never before him at the same time for comparison. He can not look from one to the other, but must carry in his memory the picture as he saw it. Patients have queer ways of comparing letters. It is not rare for one to say first way is clearer than second, and yet when he is made to prove it by reading the letters, he reads more with the second than with the first.

With either method as demonstrated in the laboratory on models with glass lenses, the differences produced by the equivalent of .25 D or .12 D astigmatism are so obvious that prompt and definite selection is easy. When the tests are applied clinically on patients who are good observers and have good eyes, the results are as prompt and definite as a knowledge of the theory would lead us to expect. In other words, when the tests work well they are very satisfactory! Before taking up the

results in the cases which I studied for this paper, it will be worth while to consider the fairly large group of cases where these tests do not work so smoothly.

In determining the focus of lenses or of eyes, we are finding the point where the rays of light meet. There are two difficulties which interfere with the tests, the first physiological, the second psychological. In the most favorable cases the meeting point is hard to locate because the lines cross at such a small angle. Any one of a series of points is so much like its neighbor as to make a choice very difficult.

There is a second and more important physiological reason for failure in finding the exact focus of an eye. The reason the patient cannot tell you the exact point where the rays meet is because there is no such point. In most cases the rays never meet at a common point. Much emphasis in elementary studies on the behavior of light rays passing through infinitely thin lenses has had a tendency for us in our thinking to picture the rays of light in the eye as meeting in a point, or in cases of astigmatism as following the conoid of Sturm and meeting in an anterior focal line, and again in a posterior focal line. Gullstrand has emphasized this point by saying that the path of the rays in the eye does not even remotely resemble the conoid of Sturm. This is misleading in the other direction, for it is only that component part of the ocular defects in refraction which is closely like the regular conoid of Sturm that we can correct with our spectacle lenses, since they have that approximate effect on the rays.

Just how the rays do behave in the eye, and just how the eyes deal with the problem in their efforts to see as well as they can is a matter about which we should have clear ideas if we are to apply any of these tests in practice.

In the first place, even if the cornea and the lens had true perfect spherical surfaces, the rays would not be refracted to a point because of spherical aberration.

In the second place, the surface of the cornea, which does the greater part of the refracting, is so far from a true sphere that there is usually a difference of as much as a diopter, and often much more, between different parts of the cornea of the same eye.

The result is that rays are rather badly jumbled together when they get to the retina. There is not a focal point, but a focal area or pattern. The rays are condensed so that all come within this area. The important thing is that they are not uniformly distributed in this area. If they were, things would be pretty bad. There are one or more parts of the pattern where the rays are more numerous. It is usually possibly for the eye to make a selection by accommodation, choosing that focal length which brings out the best of the pattern. The result may be that a point is imaged on the retina as a fairly well-defined point, but in an area or pattern the rest of which is fainter and which the observer can ignore. Anything like a smaller pupil by cutting down the brightness of this faint area which envelopes the point image helps the observer to ignore it.

If we bear in mind these facts about the way the rays are refracted by the eye, and the imperfection of the images formed, we shall be prepared to understand responses by the patient, and why it is so hard sometimes for them to give satisfactory answers.

Under psychological difficulties are to be grouped such factors as poor powers of observation, inattention, lack of interest in the test the significance of which the patient does not understand. The blurring produced by the fogging lenses is a source of trouble, because unless it is explained to the patient he is apt to think that you are trying to make the letters clear, and he has a poor opinion of your success.

Following is a summary of the comparative findings by the two methods:

DIFFERENCE IN STRENGTH OF CYLINDER FOUND BY THE TWO METHODS	DIFFERENCE IN AXIS
0.00 D in 25% of the cases	0°- in 62% of the cases
.12 D in 30% of the cases	0- 5° in 12% of the cases
.25 D in 21% of the cases	5-10° in 10% of the cases
.37 D in 10% of the cases	10-20° in 10% of the cases
.50 D in 10% of the cases	20-30° in 2% of the cases
.75 D in 2% of the cases	30-60° in 2% of the cases
1.00 D in 2% of the cases	60-90° in 2% of the cases
100%	100%

The fact that there was no difference in the axis in 62% of the cases and not over 5° difference in 74% bears out Dr.

Crisp's conclusion that the crossed cylinders are a very reliable method of finding the axis. In the majority of cases (all those who are good observers and have rather small errors) the astigmatic dial was quicker and more convincing. In a few cases when the dial was unsatisfactory because the patients' answers were either indefinite or contradictory, the crossed cylinder proved a valuable aid pointing toward the correct axis which was afterwards confirmed by the dial.

The usual procedure was to determine the axis with the dial, measure the strength of cylinder required, and then with a slight fogging (.25 D to .50 D), try the crossed cylinder.

It is only fair to say that if the order of the tests had been reversed in the majority of cases instead of in a few only, the findings would probably have favored the dial more than they did.

The cases of large discrepancy in axis were four of them in astigmatism of only 0.12 D, as measured by one or both methods. A few others were rejected because the findings were equivocal and unsatisfactory, and time could not be taken to sift out the truth.

It is reassuring that in the matter of *amount* of astigmatism the agreement between the two methods was almost as striking; not over .25 D difference in over seventy-five per cent of the cases, not over .50 D in ninety-six per cent.

The larger differences were in very high astigmatism, or in low visual acuity. On the average the dials showed about two per cent stronger cylinder than the crossed cylinders.

The shape of the letters has some effect on legibility, depending on whether vertical or horizontal lines are more distinct; hence it was expected that the crossed cylinders would show a tendency to favor less plus cylinder with the rule and more plus cylinder against the rule. This proved to be the case, but the tendency was not very marked. Taking all cases where the axis was within 15° of 90 or 180, the dials showed a stronger cylinder (more plus or less minus) in the vertical meridian in forty-one per cent; in thirty-two per cent the crossed cylinders showed a stronger cylinder needed; in twenty-seven per cent there was no difference.

It occurred to me that the use of the broken circle as a test object, or the acute angled characters of Beach, might eliminate this source of error. Carefully made photographs showed that

with the weakest crossed cylinder recommended by Dr. Crisp, viz: + .25 ⊃ — .25, distinction between the two positions of the cylinder was very easy to make with either letters or other symbols. This is an illustration of the superior refinement of the dials. In the final test with the astigmatic charts the patient is expected to respond to a cylinder of .12 D held first at one axis and then at the other. If he does so, this shows that the cylinder he is wearing is correct ±.12 D. With the crossed cylinder test the weakest cylinder recommended is + .25 ⊃ — .25, or a total cylindrical power of .50 D. With this the patient is expected to respond by telling which position is better, with or against the cylinder he is wearing; and he is usually able to do so promptly and consistently. This shows that the cylinder is correct ±.50 D. If now the cylinder in the trial frame is increased or decreased .12 D, and the crossed cylinder again tried, the decision becomes: which is better, adding .50 cylinder to the .12 already added, or subtracting .50? If the patient responds correctly, this test is also correct ±.12.

The impression left in my mind by the comparison is that both tests are valuable, but that when the best models are used the dials give more prompt, consistent and convincing results in a larger percentage of cases than any other test for astigmatism. There is a minority of cases, however, where the dials do not yield prompt and consistent results because of irregular astigmatism or poor powers of observation, but where the crossed cylinders are more convincing and consistent. Often in these cases a second trial with the dials after the crossed cylinders have been used, will confirm the latter and leave a feeling of conviction in the examiner that he has the correct result.

None of the tests can be expected to give the best results without long practice and study. Above all, do not judge any method by the results obtained when the exact technique has not been followed.

DISCUSSION

Dr. Edward Jackson, Denver: Scientific progress surely is in the making when a paper of this character is placed on the program of the Academy. Those of us attending see new apparatus and instruments presented, but as is usual with new instruments, they refer to operative ophthalmology. Certainly good progress in ophthalmology can only be

made in a study of the causes of disease and the refinements of methods of control. This subject under discussion has that especial significance.

Perusing the title I was inclined to take issue with a paper that should urge one test against another. Hearing Dr. Lancaster's paper, particularly his conclusions, I agree with the general trend of his argument; reserving, however, the privilege of discussing the points used in the comparison of the tests.

I have used a revolving chart with three radiating lines, first using the line that could be distinguished at thirty feet, then one for twenty feet, and finally using at twenty feet a chart with lines that were only distinguishable at fifteen feet, as a test of astigmatism. These are subjective tests, in which the psychology of the patient is made use of. There are subjective tests that are capable of practical objective confirmation, and one of these is the cross cylinder test. Another test is the confrontation test for taking the fields of vision. I myself never take fields of vision without first testing the patient in this way. This test may be made objective by moving the fingers, or holding them still, asking the patient to say whether they are moving, or still. If the sense of motion is definite in the peripheral fields it becomes in effect an objective test.

With the cross cylinder the objective certainty can be reached in a different way. Try to focus the meridians with the cross cylinder, and when you think you have them focussed, change the cylinder and try the patient again. In this way I test the meridians for astigmatism three to six times at one sitting. The patient knows nothing about how you are turning the cylinder, does not know where the plus axis or the minus axis is, has no guide to decide where he should want the cylinder in the trial frame, obviating the occasional ambition on the part of the patient to help. When you secure a half dozen such tests agreeing within one degree of each other, reliance can be put upon the chosen axis for the cylinder with more certainty than upon the ophthalmometer, or the shadow test methods, which are purely objective. The same certainty is reached when the method is modified to get the strength of the cylinder required. I very frequently find that patients, repeatedly tested, prefer something stronger than one diopter cylinder, and something weaker than one and a quarter cylinder. It is a much more accurate way of deducing intervals of one-eighth than to use cylinders that change by one-eighth diopter. By reasoning and repeating our tests, the cross cylinder can be made to have the value and force of an objective test. It is not simply that the eye is an imperfect optical instrument, we must recognize the psychic element in vision, which is very important.

The important thing in subjective tests is to master and make use of the psychology of the patient, in order to insure accuracy. Often times this requires repetition of tests. Through repetition the cross cylinder will permit an accuracy that cannot be reached by cycloplegics, or with the ophthalmometer, or with the most accurate objective tests.

In the first twenty years in which I measured refractions I used some type of a rotating chart; but finally came to the conclusion that with the ophthalmometer, shadow test, and then the cross cylinder, and with the

eye under a cycloplegic and post-cycloplegic testing my results were the best.

Dr. S. Judd Beach, Portland, Maine: These two methods of examination which have been discussed are not mutually exclusive. Neither can be neglected if extreme accuracy of refraction is desired. Disappointment in these devices may be in part due to lack of understanding of their limitations. The astigmatic dial is unsatisfactory in irregular astigmatism. Some years ago I studied the appearance of the astigmatic chart as described by a series of patients, and found that a misleading degree of irregularity is unexpectedly prevalent. One type of asymmetry, for instance, will place the astigmatic axis in the upper hemisphere twenty to forty degrees from that below the horizontal. Such a possibility ought entirely to preclude the use of the half dial or astigmatic fan which gives no clue to this discrepancy.

The weakness of the cross cylinder lies in the fact that it has to be used in connection with some form of test card. Patients often prefer a glass that somewhat distorts certain letters by over-emphasizing distinguishing lines. They are also confused by the difference between our test type and their ordinary printing, which is higher than wide and has the vertical lines accentuated. Moreover, as I have frequently pointed out, the acute-angled letters are a more sensitive test for astigmatism than round ones and those with right angles.

If trial characters for the cross cylinders are intelligently selected, and if too much reliance is not placed on the astigmatic chart in cases of irregular astigmatism, these two tests take their place among the most delicate and useful that we have.

Dr. Walter B. Lancaster, closing: I wish to thank Dr. Jackson and Dr. Beach for adding so much to the paper by their discussion.

I think if anyone wishes to excel in measuring refraction he should try these tests on himself to understand just what happens when certain changes are made either in sphere, cylinder, or axis. These, combined with the study of the psychology of the patient, will help us a good deal to overcome the lack of knowledge of physiological optics, and especially visual optics.

Dr. Jackson pointed out one thing which I think explains why I find I get along faster with dials than cross cylinders. He says he takes four or five or a half dozen readings and averages them. With the dial you do not do that. If the case is at all suited to the dial and the patient picks out certain lines, you find the center of that group of lines; this is the axis; then measure the strength of the cylinder required by shifting the power of the cylinders until the two lines on the second chart are equal. The final test of accuracy is putting a plus or a minus 0.12 cylinder over the cylinder in the frame parallel to its axis, and the same cylinder the other way at right angles to the axis. If the patient detects which way you place it this shows you are right within ⅛ diopter.

In getting the axis of the case Dr. Beach pointed out, where the meridians do not seem to agree, I think the best guide is this: Having gone over the case and arrived at a conclusion as to the approximate strength of the cylinder, put in a sphere just equal to the strongest meridian. You thus reduce your fogging to a minimum, and in that way you will very frequently get a fairly satisfactory and convincing axis. This should then be confirmed by the cross cylinders.

DETACHMENT OF THE RETINA; THE GUIST OPERATION

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GONIN startled the ophthalmological world in 1927 and 1928 with his reports of favorable results in the surgical treatment of detachment of the retina by use of the thermo-cautery. We have used the Gonin procedure but with no such high percentages of cures and an inquiry sent to a number of ophthalmologists was answered with very discouraging reports. Several oculists wrote in detail to tell of their one or two cures but no one claimed that their combined cures and improved cases amounted to more than ten to fifteen per cent.

A great many articles have been written in the past three years describing the work done to improve on the treatment of detachment of the retina. In this country Dr. Mark J. Schoenberg of New York has contributed valuable information in reporting results of operations he performed by the Gonin method. He has pointed out that the success of the procedure depends on (1) the selection of suitable cases, (2) the accuracy of the technique, and (3) the cooperation of the patient.

In 1931 Dr. Leo L. Mayer of Chicago reported results and conclusions of experimental study of detachment of the retina. Two very valuable points in his conclusions are, that although reattachment occurs when the cautery is used, there is marked destruction of tissue in the vicinity of the operative wound; and that methods that do not cause this extensive destruction, but at the same time, produce an exudate that seals the retina to the choroid, would seem to be those of preference. This latter point is demonstrated in Fig. 1, which shows a continuous layer of retina reattached and yet not destroyed. The method of Guist was used, the stick being applied for one second.

In 1931 Dr. J. Ringland Anderson published a monograph which is a beautiful contribution to the study of the causes and treatments of detachment of the retina. The history of retinal

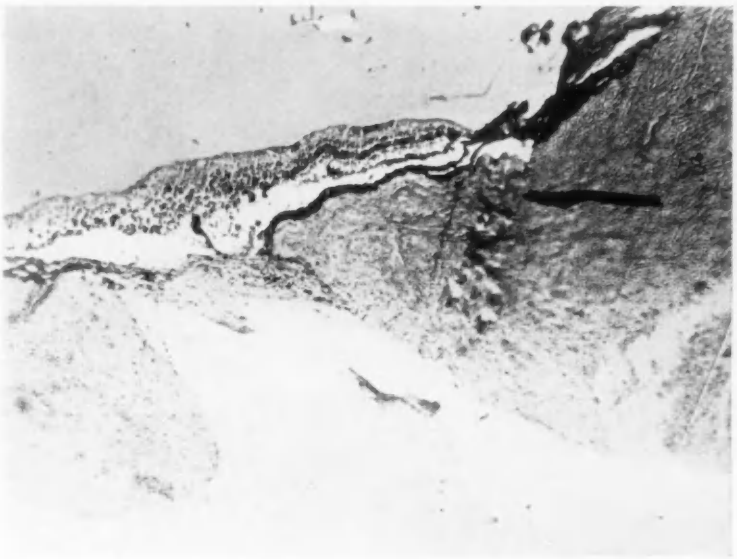

FIG. 1 — (McKeown) — After Mayer. Reattachment of artificially detached retina treated for one second by potassium hydroxide stick.

detachments as he indicates has been a perplexing problem for many years and the infinite detail with which he has described the pathology in these cases is to be commended. In his closing chapter the Gonin method of surgical treatment for detachment of the retina is recommended as the best. This is followed by the statement that Gonin, Vogt and a few others, expert in the technique of the ignipuncture, have claimed approximately fifty per cent of cures.

A brief review of the histology and pathology of the detachment of the retina at this time further augments the advantages to be gained in the use of the method we are about to describe.

Anderson points out that the invagination of the primary optic vesicle leads to the formation of two layers, which later come into contact but do not unite. It is this line of contact which becomes a line of cleavage in the development of a retinal separation or detachment. This line appears to be a developmental weak spot. In early fœtal life it is a ciliated space. The cilia disappear from the inner surface of the optic cup as the pigment granules appear, while the cilia of the opposing surface

of the inner wall probably increase in size as they diminish in number. This is the formation of primitive limbs of rods and cones which early appear to adhere to the pigment epithelium. This attachment throws light on the adherence of these two layers when the mature retina becomes separated or detached by artificial or morbid conditions.

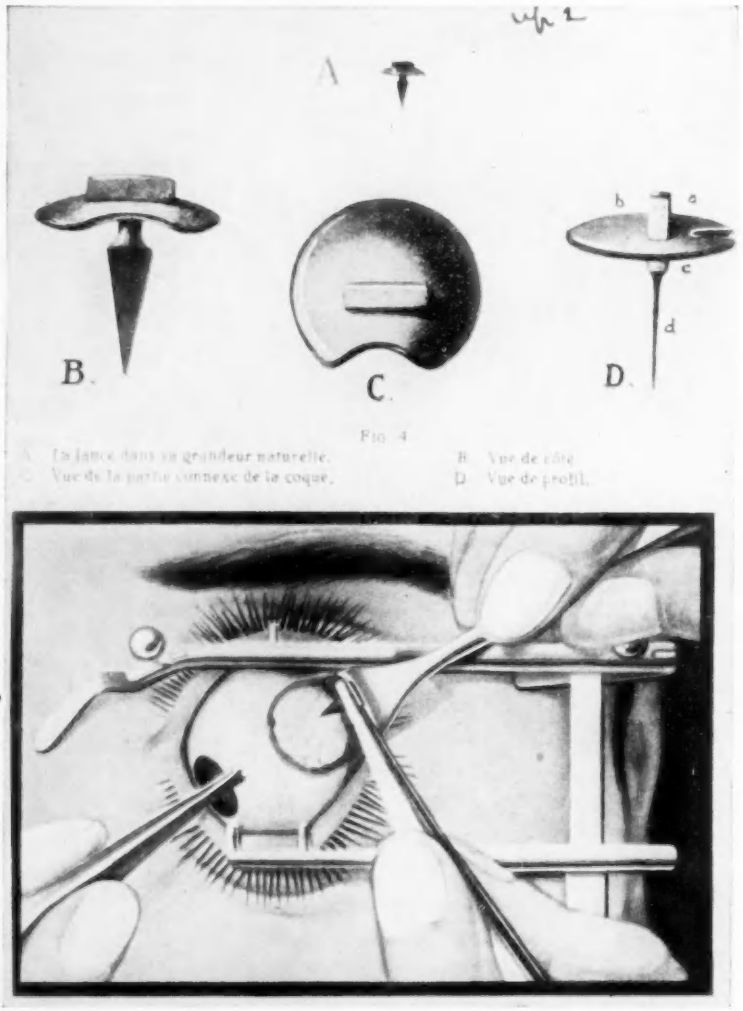

FIG. 2—(McKeown)—Lancet of H. Arruga used for localizing a tear in the retina.

In choroidal disease the pigmented epithelium degenerates and it is noticed that the neuro-epithelium is also affected and that the pigment migrates. This migration and the ability of the lamina vitrea to proliferate probably aid the reattachment of the retina when it is reapplied. With this knowledge we believe that better results will be obtained when the separated layers of retina are brought together and adhesions formed in several areas.

Dr. H. Arruga of Spain has recently reported and published his results of the study of 216 cases of which 164 were operated. He reports fifty-three cures, which is thirty-three per cent of all cases operated. He used the Gonin ignipuncture, the Galvano-cautery, the Guist operation and in one case a combination. Dr. Arruga classifies his cases as easy, difficult and very difficult. Under this classification he reports fifty per cent cures in the easy cases, ten per cent cures in the difficult cases and no cures in the very difficult cases.

Dr. Arruga advanced a theory on the origin of subretinal fluid. He stated that the choroid and vitreous are incompatible organs and that when they come in contact a reaction occurs. He further believes that a chorio-retinitis causes the hole or holes in the retina and that the choroid attracts and absorbs fluid vitreous, drawing it through the retinal hole producing the increased subretinal fluid. When the hole or holes are closed there is no more accumulation of fluid behind the retina.

Localization of Detachment and Tears

Dr. Gustav Guist has constructed an ophthalmoscopic localizer (Fig. 3) and with this instrument an accurate outline of part of the detached retina may be obtained. Also the exact position of the retinal hole or holes may be located and indicated upon a chart (Fig. 4) diagrammatically arranged to be used at the time of operation.

There is also a small instrument which Dr. Guist calls a "Schema" (Fig. 5) which is sutured to the sclera covering the cornea. The arms of the instruments are cut in known lengths and are used to measure the location for the scleral trephining. This instrument is removed after the scleral impressions have been made with the trephine. A one and one-half millimeter trephine is used. The Guist lid retractor is used to advantage as it lifts the lids from the globe which becomes hypotonic dur-

FIG. 3 — (McKeown) — Guist Detachment Localizer.

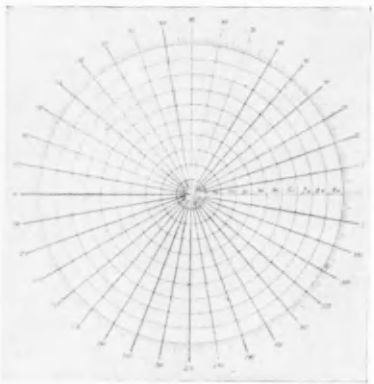

FIG. 4—(McKeown)—Chart for recording.

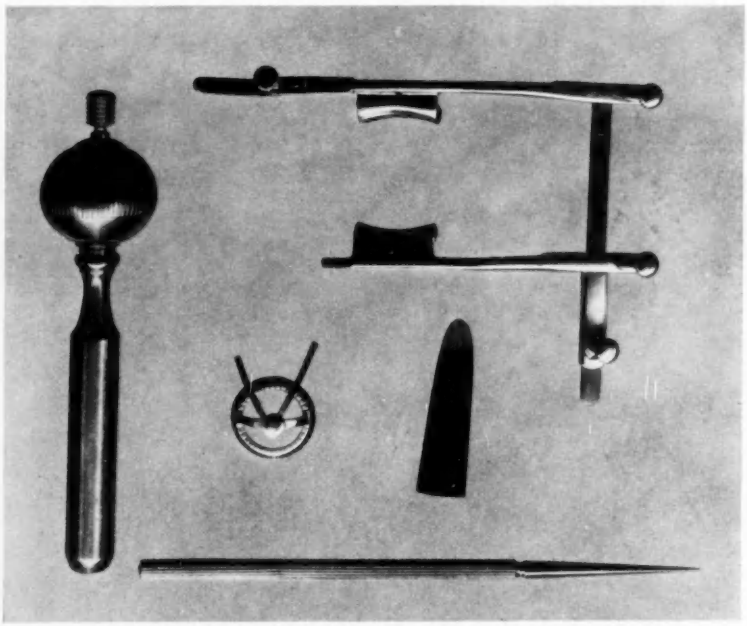

FIG. 5—(McKeown)—Guist Speculum, Schema, Probe and KOH Stick.

ing the operation. Fig. 5 also shows a paraffin covered stick of KOH and a form used in arranging and soldering the arms of the "Schema."

Technique of the Operation

The pupil should be well dilated with atrophine and adrenalin, if necessary, and the eye anesthetized with two per cent holocaine solution. Two per cent novocaine solution injected into the recti muscles adjacent to the field of operation will augment anesthesia and allow greater rotation of the globe. A long incision is made in the conjunctiva over the insertion of a rectus muscle parallel with the limbus of the cornea. A double armed silk suture is put in the muscle which is cut close to the insertion. Then with another suture in the stump of the muscle the eye may be rotated, giving the desired exposure of the sclera. It is at this point that the "Schema" is used and impressions made in the sclera with the trephine, outlining the area to be operated.

The trephine holes through the sclera are carefully completed using the trephine and a keratome or Ziegler knife in a

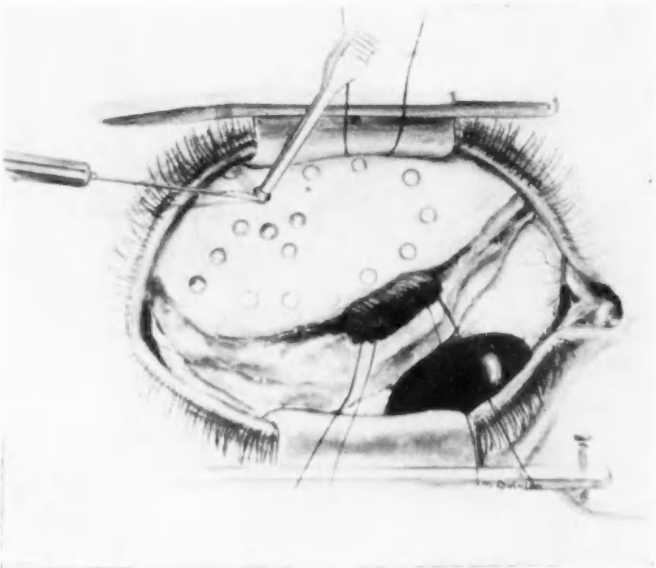

FIG. 6—(McKeown)—Case 10—Trephine impressions and removal of scleral plugs.

shaving manner, removing the inner fibres of sclera and exposing the choroid, care being used not to cut choroidal vessels. The number of trephine openings depends on the extent of the detachment and the number of holes in the retina (Fig. 6).

Through each of the scleral openings the choroid is treated with potassium hydroxide in the form of small paraffin covered KOH pencils, the paraffin being removed from the tip just before application, as caustic potash is extremely hygroscopic and immediately melts in the air. The point is applied to the choroid for two or three seconds whereupon the spot is directly neutralized with one-half per cent acetic acid by means of a swab and irrigated with normal saline. The field must be dried and the caustic point dried before each treatment to prevent too severe cauterization. When all of the trephined areas have been cauterized and neutralized, care must be taken in every case that none of the wound edges of the trephined sclera bleed.

The choroid is then perforated with a small blunt sound or punctum dilator thereby opening the subretinal space at every trephined and treated area (Fig. 7). All areas need not be perforated if the surgeon believes sufficient drainage has been in-

FIG. 7—(McKeown)—Case 10—Exposed choroid and treated areas. Release of subretinal fluid.

stituted. The muscle is reattached to its stump and the conjunctiva closed in the usual manner. Binocular dressing is applied. The patient should rest quietly in bed for several days.

We have performed the Guist operation on sixty cases since September, 1931. Nine of the cases are so recent that we cannot report the result at this time. Of the other fifty-one cases I have tabulated certain important observations which give us information as to what result might be obtained in the use of this method. I have classified the patients as cured, improved and unimproved. I designate as cured the cases in which the retina has been reattached and in which the visual function has been restored to 20/100 or better. The improved cases are those in which the retina has been replaced but the visual function has not been restored.

Of the fifty-one cases reported, there were twenty cures and seven improvements.

Cures	20	39%
Males	32	63%
Females	19	37%
Right eyes	29	57%
Left eyes	22	43%
Myopia	32	63%
Hyperopia	3	5%
Emetropia	11	21%
Aphakia	5	10%

In three cases in which the Gonin procedure had failed the retina was reattached by the Guist operation. Complications which have occurred in my cases are intraocular hemorrhages in five cases, endophthalmitis in one case, and hypotony in three cases which continued four or five months, probably due to cyclitis. By operating with great care these untoward results may be kept to a minimum.

Report of Cases

Case 1—L. B., age 40. Complete detachment of retina, right eye. Gonin operation, 6 months previous. Guist operation produced reattachment of retina but no improvement in vision.

Case 2—M. J., age 62. Detachment upper temporal field, left eye. Guist operation, August 25, 1931. Partial reattachment which held 5 months. Patient not seen since.

Case 3—E. C., age 28. Myopia. Sudden detachment of retina, left eye, five days previous; result of a fall down stairs. Guist operation September 29, 1931. Reattachment of retina below. Subsequent detachment above, 3 months later complete detachment.

Case 4—H. H., age 26. Myopia. Struck in right eye by boxing glove six weeks previous (Fig. 10). Guist operation October 9, 1931, resulting in perfect peripheral field and normal vision which has been retained for 11 months.

Case 5—J. B., age 39. Myopia. Loss of vision, right eye, following football scrimmage. Guist operation complicated by loss of fluid after first trephine. Two reoperations failed to reattach retina.

Case 6—D. S., age 53. Myopia. Right eye, retina had been detached 10 years. Recent detachment of retina of left eye following blow on head. Guist operation October 23, 1931. Retina attached and subsequently detached 2 months later. Second Guist operation did not reattach the retina. Gonin operation attempted resulting in intraocular hemorrhage. Absorption of hemorrhage is not complete.

Case 7—R. H., age 8. No refractive erorr. Struck in right eye by tennis ball thrown about 20 feet. Retina torn from ora serrata inferiorly (Fig. 11). Guist operation October 27, 1931, resulted in reattachment of retina and normal vision which has been retained to date.

Case 8—P. L., age 41. Myopia. Detachment of retina of right eye for 3 weeks. Denied recent injury. Guist operation, October 30, 1931. Retina reattached and detached 6 weeks later. Second operation December 11. Retina reattached, which held for 3 months.

Case 9—M. P., age 36. Myopia. Sudden impairment of vision of right eye in March, 1931. Guist operation October 10, 1931, resulting in a good field and 20/50 vision. Retina has remained attached to date.

Case 10—R. C., age 45. No refractive error. Struck in right eye by child's hand. Detachment in upper temporal quadrant (Fig. 8). Guist operation December 11, 1931. Eighteen 1½ mm. trephines (Figs. 6 and 7). Two months later (Fig. 9)

the retina was in place and the vision without correction was 20/25. The retina has remained in place to date.

Case 11—S. N., age 36. Myopia. Sudden loss of vision, right eye, while lifting heavy crates. Guist operation December 24, 1931, reattached the retina and improved the vision until February 13, 1932, when retina again became detached. Second Guist operation was not successful.

Case 12—F. A. W., age 30. Myopia. Hemorrhage of right eye, 2 months previous, resulted in detachment of the retina in lower half. Guist operation on January 7, 1932, resulted in extensive intraocular hemorrhage and ultimate endophthalmitis.

Case 13—S. Z., age 55. Myopia. Loss of vision of left eye about December 20, 1931. Vitreous was turbid, indicating intraocular hemorrhage. The retina was elevated in the upper temporal quadrant. Patient was observed for 5 weeks and then the Guist operation performed. Patient was discharged from the hospital and the first follow-up visit revealed new intraocular hemorrhages.

Case 14—S. M., age 40. Myopia. Poor vision, right eye for 3 weeks. Retina detached in lower field. Guist operation reattached retina for 3 weeks only. To be reoperated.

Case 15—J. Mc., age 47. Myopia. Poor vision in right eye for 7 weeks, resulting from foreign body. Guist operation failed to reattach retina.

Case 16—B. H. F., age 63. No refractive error. Detachment of retina, left eye for 1 month, following head hitting top of automobile after driving over a bump. Guist operation failed to reattach retina.

Case 17—L. W., age 36. Myopia. Detachment of retina in lower half of right eye for past 6 weeks. Guist operation February 16, 1932, reattached the retina and restored vision which has stayed to date.

Case 18—J. J. B., age 46. Myopia. Detachment of the temporal half of retina, left eye, for 4 months. Had been resting in bed in hospital most of time. Guist operation February 25, 1932, reattached retina and restored vision to 20/70 +. Unchanged to present time.

Case 19—S. S., age 18. No refractive error. Detachment of retina, right eye, lower temporal quadrant for 1 year (Fig. 14). Guist operation March 1, 1932, reattached the retina and improved the vision to 20/100 which has remained so to date (Fig. 15).

Case 20—S. P., age 21. Aphakic eye which had had a linear extraction of cataract 18 months previous. Detachment of retina in lower half of left eye for 8 months. Guist operation March 22, 1932, reattached retina producing a normal field and 20/50 vision with correction.

Cases 21 and 22—J. B., age 35. No refractive error. A female with a detachment of lower half of retina in both eyes. Detachment, right eye, present 4 years. There was also a hole in the macula. Guist operation, left eye, on March 31, 1932, and right eye, April 7, 1932. Result—reattachment of retina of both eyes, obtaining normal peripheral vision, 20/200 vision in right eye and 20/25 in left.

Case 23—A. B., age 26. No refractive error. For 1 week patient had had a detachment of retina in lower temporal quadrant of left eye (Fig. 16). Guist operation April 5, 1932, resulted in reattachment of retina (Fig. 17) and restoration of normal vision.

Case 24—B. S., age 12. Myopia. Detachment of retina of nasal half of right eye for 1 week, resulted from slipping on oil and falling to a sitting position. A Guist operation was performed April 7, 1932, resulting in reattachment of retina and restoration of 20/40 vision.

Case 25—H. E., age 35. Myopia. Detachment of retina, lower half of left eye for 5 or 6 weeks following a blow on the side of head. Guist operation, April 22, 1932, resulted in reattachment of retina.

Cases 26 and 27—A. W., age 25. No refractive error. A female with detachment, lower half of retina, both eyes, since May, 1931. There was a large bullous detachment of right eye (Fig. 18). The fields of this eye before and after operation are shown in Figs. 20 and 21. Guist operation of right eye on May 4, 1932, resulted in reattachment of retina (Fig. 19). This eye had had 2 previous Gonin operations. The left eye had had four Gonin operations which failed to reattach the retina. Guist

operation May 27, 1932, reattached the retina and improved both the field and vision.

Case 28—D. S., age 57. Myopia. Spontaneous detachment of temporal half of retina, left eye, 2 weeks previous to examination. Guist operation June 8, 1932, failed to reattach the retina.

Case 29—A. McG., age 26. Myopia. Detachment of temporal half of retina, right eye, resulted from bumping head on the wall 10 days before. Guist operation May 20, 1932, resulted in reattachment of retina and restoration of normal vision and field.

Case 30—J. L. D., age 62. Aphakia. Detachment of retina of upper temporal quadrant of left eye for 4 days. Guist operation, June 16, 1932, reattached retina and produced normal field and vision.

Case 31—J. A., age 24. Myopia. Complete detachment of retina left eye. Guist operation June 17, 1932, by making 9 trephines in upper half and 10 trephines in lower half. Resulted in reattachment of lower half and partial attachment of upper half, improvement in field of vision, and increase of vision to 20/200.

Case 32—N. B., age 50. Myopia. Detachment of retina of right eye in lower temporal quadrant and a turbid vitreous resulted from an automobile accident 2 months previous. Guist operation June 20, 1932, resulted in no improvement.

Case 33—G. M., age 49. Myopia. Detachment of the retina lower half of left eye for 6 months, result of being struck in eye with a stone. Guist operation June 30, 1932, failed to reattach retina.

Case 34—F. W. P., age 44. Myopia. Spontaneous detachment of retina upper temporal quadrant of left eye for 2 weeks. Guist operation, July 1, 1932, resulted in intraocular hemorrhage. As hemorrhage absorbed it was determined that retina was reattached above but had become detached below.

Case 35—G. C., age 39. No refractive error. Detachment of retina lower half of right eye. Guist operation, July 5, 1932, failed to reattach retina.

Case 36—H. H., age 29. No refractive error. Detachment of retina lower half of left eye for past 2 months. Guist operation, July 7, 1932, failed to reattach retina. Patient very uncooperative.

Case 37—W. Q., age 21. Myopia. Sudden loss of vision of left eye 6 months previous. Vitreous turbid, appearance of organized blood under detached retina. Guist operation, July 12, 1932, failed to reattach retina.

Case 38—C. D. G., age 57. Myopia. Detachment of retina lower half of right eye for 5 weeks, resulting from kick in eye by a baby's heel. Guist operation July 14, 1932, replaced the retina and restored vision. Two weeks after the patient was discharged from the hospital, retina became redetached.

Case 39—G. B., age 11. Myopia. Complete detachment of retina left eye for 4 days resulting from being knocked down by a bicycle. Guist operation July 28, 1932, failed to reattach retina.

Case 40—C. B., Jr., age 19. Myopia. Detachment of retina upper temporal quadrant of left eye following spring board diving. Guist operation on August 4, 1932, resulted in reattachment of retina and restoration of vision and field.

Case 41—H. R., age 44. Myopia. Spontaneous detachment of temporal half of retina left eye. Guist operation resulted in reattachment of retina and improvement of field and vision.

Case 42—B. F., age 52. Myopia. Detachment of nasal half of retina right eye. Resulted from a fall when alighting from a street car 6 weeks previous. Guist operation resulted in reattachment of retina and restoration of normal field.

Case 43—T. A., age 26. No refractive error. Detachment of retina upper temporal quadrant of left eye for 2 months. Spontaneous. Guist operation August 17, 1932, resulted in reattachment of retina.

Case 44—V. L., age 42. Aphakia. Detachment of retina left eye in lower temporal quadrant 4 years previously after high diving. A traumatic cataract occurred at the same time. Guist operation reattached retina but failed to restore vision.

Case 45—B. A., age 26. Myopia, aphakia. Detachment in lower half of right eye for 2 months. Patient was a bleeder.

After a Guist operation there was an extensive intraorbital hemorrhage. Retina appears to be in normal position but vision has not been improved.

Case 46—S. S., age 29. Aphakia. Detachment of retina lower half of right eye, resulting from rough treatment by "hold-up" men 2 months previous. Guist operation resulted in reattachment of retina but no improvement of vision to date.

Case 47—I. B., age 61. Myopia. Detachment of retina upper temporal quadrant right eye for 5 weeks. Guist operation reattached the retina and restored normal peripheral fields and 20/100 vision.

Case 48—M. W., age 33. Myopia. Complete detachment of retina left eye for 7 weeks spontaneous. Guist operation failed to reattach retina.

Case 49—J. T., age 51. Aphakia. Detachment of retina lower half of left eye. Guist operation replaced retina and improved field but to date vision has not been improved.

Case 50—C. R., age 70. No refractive error. Detachment of retina of left eye for 8 years. Detachment of lower half of retina right eye for 1 year. Guist operation reattached retina of the right eye, restored the field, but only slightly improved visual function.

Case 51—R. M., age 28. No refractive error. Three months pregnant. Detachment of retina lower temporal quadrant left eye (Fig. 12) for 6 weeks. Guist operation replaced retina, restored field and vision, but hypotony has persisted for 2 months.

FIG. 8—(McKeown)—Case 10—Bullous detachment before operation.

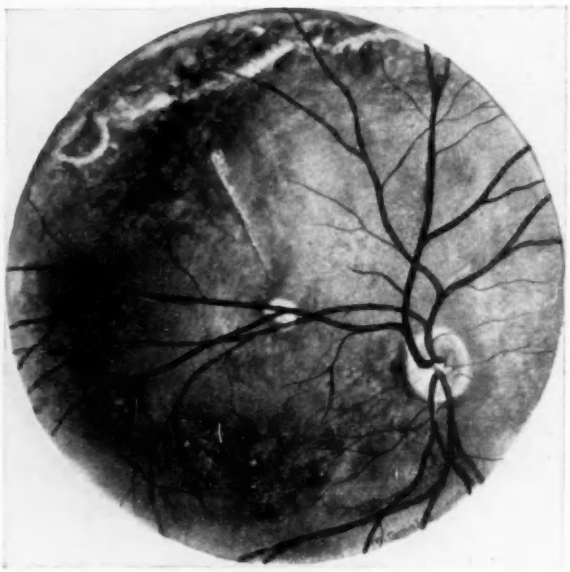

FIG. 9—(McKeown)—Case 10—Reattachment of the retina after operation.

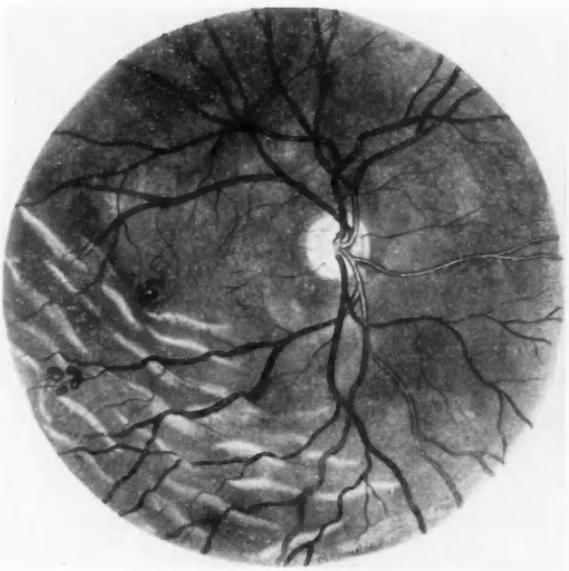

FIG. 10—(McKeown)—Case 4—Flat detachment before operation.

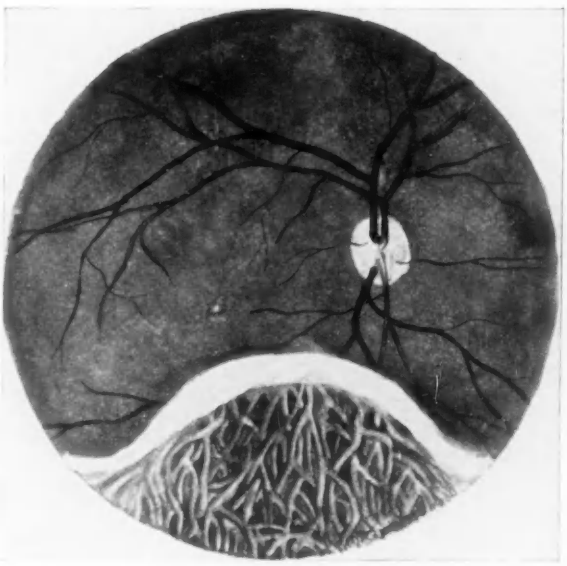

FIG. 11—(McKeown)—Case 7—Disinsertion before operation.

FIG. 12—(McKeown)—Case 51—Bullous detachment of retina with wide separation from ora.

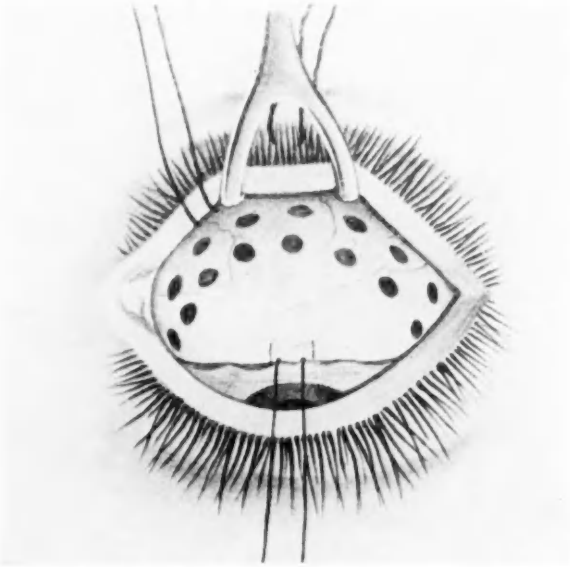

FIG. 13—(McKeown)—Scleral trephinations—Lindner.

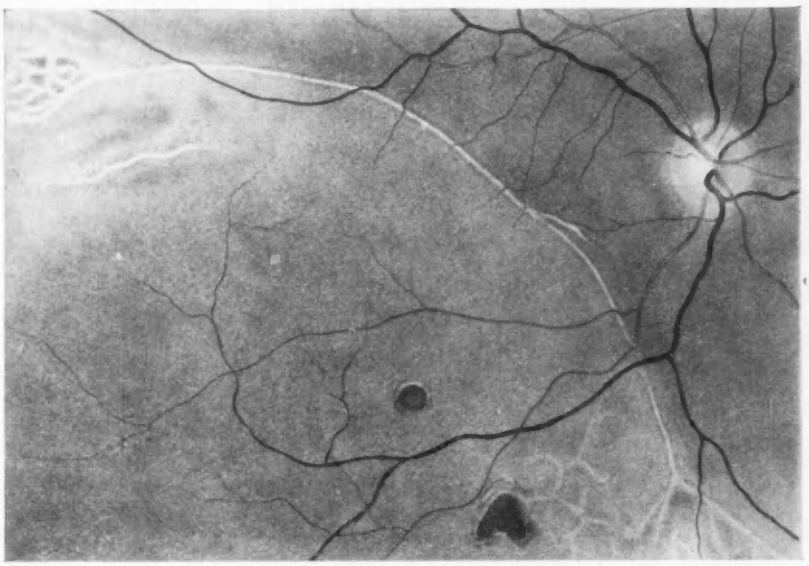

FIG. 14—(McKeown)—Case 19—Flat detachment before operation.

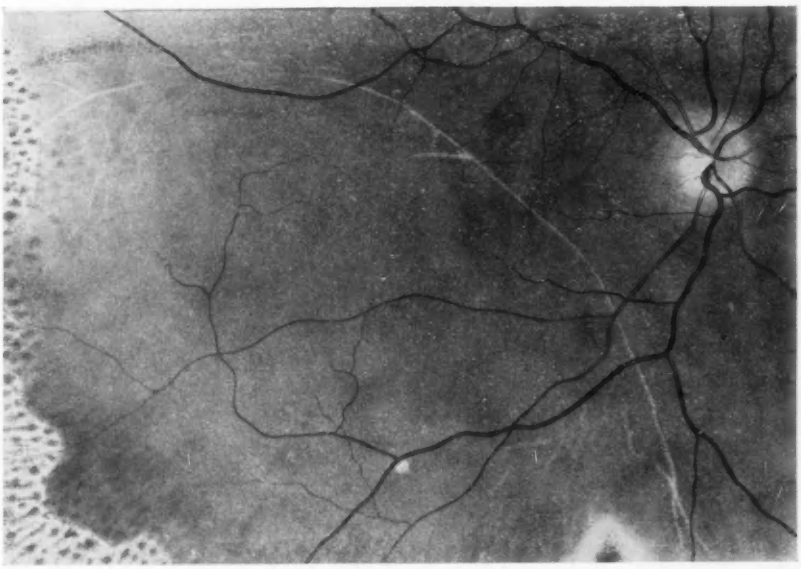

FIG. 15—(McKeown)—Case 19—After operation.

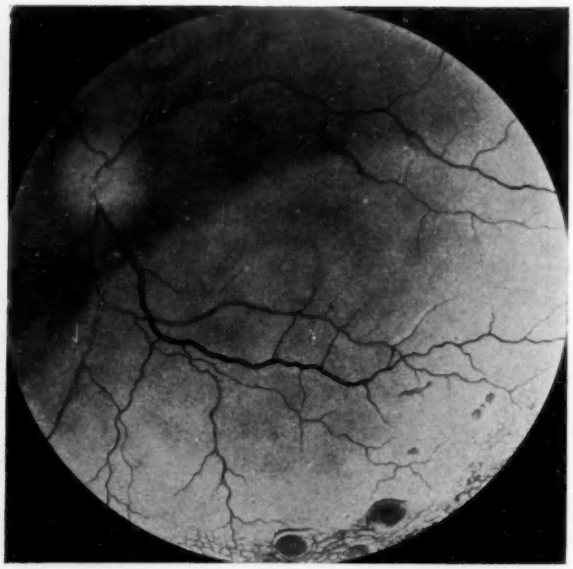

FIG. 16—(McKeown)—Case 23—Flat detachement before operation.

FIG. 17—(McKeown)—Case 23—After operation.

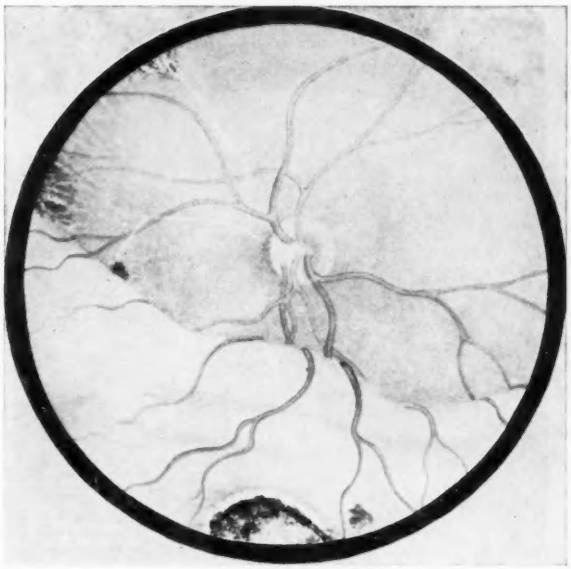

FIG. 18—(McKeown)—Case 26—Bullous detachment and cicatrix result of Gonin operation.

FIG. 19—(McKeown)—Case 26—After operation.

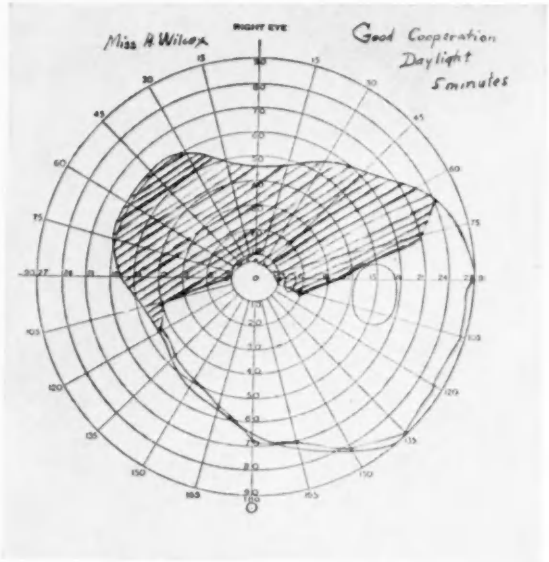

FIG. 20—(McKeown)—Case 26—Field of vision before operation.

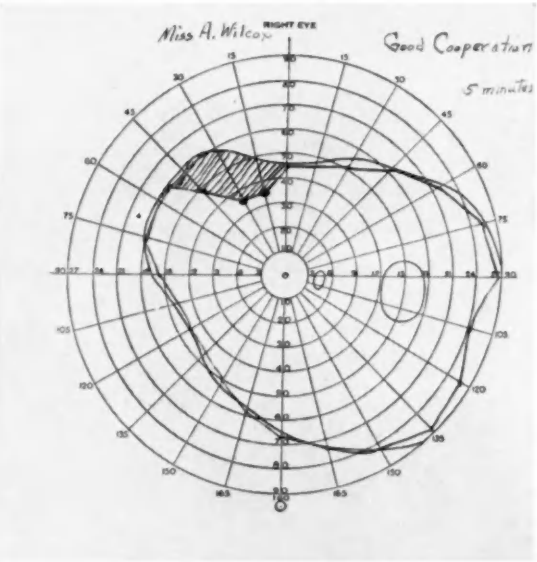

FIG. 21—(McKeown)—Case 26—Field of vision after operation. Note scotoma probably due to Gonin operation cicatrix.

DISCUSSION

Dr. Harry S. Gradle, Chicago: Again I am pinch-hitting, taking the place of Dr. Schoenberg, who was to have opened the discussion of this paper.

Dr. McKeown has presented a splendid paper and has covered the subject thoroughly. The only addition I would recommend would be a more detailed analysis, which will undoubtedly appear in the published paper. It is so good that I would like to have his results at the end of one year, and if they are as good as they are now I shall go to New York to learn the method.

The end results of various operations should be made public only after nine to thirteen months with a comparison of results of the condition at the end of six weeks, at the end of nine months, and then fifteen months, we can formulate an opinion. There have been only two sets of statistics published of cases that have been operated fifteen months or more. It would be interesting to find out what figures do prevail. We have had less than fifteen per cent good results over a long period of time, and from what I have seen of other tables, I am sure that if fifteen per cent of good results in detachment of the retina by surgical methods can be maintained we can honestly congratulate ourselves since that is nearly fifteen per cent more than by any other method. Credit is due to Gonin more for having brought the subject up to date than for a method of curing detachment of the retina.

To my mind the etiological importance of the hole in the retina is questionable. There are undoubtedly numerous cases of detachment of the retina that have become reattached spontaneously despite the presence of a hole. There are undoubtedly numerous cases of detachment of the retina in which there is no hole whatever. Of course when we say "hole" we have reference rather to the smaller tears in the retina than to the larger rents proceeding posteriorly from the ora serrata.

The operations I have tried are the Gonin, the Guist, and a trephine of the sclera with diathermy through the trephine aperture to the sclera. I do not think that any one type of operation is suitable in all cases, any more than any one type of operation is suitable for all cases of glaucoma. The older methods of diathermy have been in my hands completely unsuccessful. The newer methods of diathermy I shall not mention since Dr. Kronfeld, who has just returned from an extended stay abroad, has consented to discuss them this afternoon.

In certain cases of detachment of the retina the original Gonin method is entirely unsuitable. In case of a limited detachment of the retina, with or without a hole that can be localized, the Gonin offers a certain percentage of chance for a good result; but there may exist detachment of the retina in an area where another technic might be more successful. Perhaps here the Guist operation would be indicated, but, frankly, I am afraid of the Guist proceedure. I am afraid of a cautery that I cannot control; I am afraid of possible drainage of the eyeball; I am afraid of the large choroidal and sub-choroidal hemorrhages that so frequently occur during this operation.

I have just one other point to offer. It is easy to perforate the choroid, and usually when that is done the operation is concluded for the day. Instead of trephining for the Guist, or the galvanic cautery, or whatever method is intended, I am suggesting a more simple method. After exposing the area in which I wish to make the aperture through the sclera I incise the sclera with a flat scalpal, the same incision as is used in cyclodialysis; then I form a pocket 5 mm. in depth and 2 or 3 mm. each side, separating the choroid from the sclera. Into the pocket thus formed I insert the flat blade of the Herbert punch forceps and bite out a scleral button. Instead of a round trephine aperture, the excised portion of sclera is triangular, with a slightly more ragged aperture than the trephine.

I believe we are just at the beginning of the work in detachment of the retina. We have been groping around blindly in the dark, not knowing exactly what we are dealing with, and trying to find some method of securing the retina to the choroid. So far as refinements of technique of localization of tears go, seemingly we are agreed, but we are working in three dimensions and we can only localize in two dimensions. The illustration Dr. McKeown showed of a large, bullous-like detachment in the lower quadrant demonstrates that it is nice enough to localize the hole according to the meridian and the apparent distance from the disc, or ora serrata. But when the subretinal fluid is evacuated the retina drops back, in which case we will not know within two or three millimeters where the hole is located, and if we strike the hole accurately, luck must be given some credit.

Dr. Daniel B. Kirby, New York: The value of the Guist procedure as practiced by Dr. McKeown is that it gives a method of attack on the entire area of detachment of the retina. The sealing of any hole or tear which may be present to prevent the herniation and drag of the vitreous on the retina is important. This may be accomplished with a certain degree of good fortune by the Gonin procedure. It is accomplished with greater ease by the Guist method, which secures in addition cohesion of the reapposed retina over the entire area of detachment. It is not necessary to localize so accurately the tear or hole, as a number of trephine openings may be placed at or near the area of the sclera to which that portion of the retina will float when the subretinal fluid is released.

There is great importance in the matter of accuracy of the trephining technique, so that puncture will not be made and fluid allowed to escape and intraocular tension lowered before the trephinings are completed. One must select a good sharp instrument and then proceed cautiously until he gets the feel of the instrument.

I have two cases amongst others which are interesting to report.

A girl of eighteen years had been hit in the right eye with a tennis ball two years before her visit to me. Detachment occurred fifteen months after the injury. Her vision was 10/200, with central scotoma and a large field defect corresponding to a detachment of the retina inferio-temporally. On March 1, 1932, she was given avertin-ether anaesthesia and the Guist procedure, employing twenty-eight trephine openings was done with Dr.

McKeown's cooperation. The subretinal material was fluid. The vision is now 20/70 with complete reattachment of the retina. There remains a small central scotoma and a peripheral field defect which is less than it had been. Her intraocular tension is now right 8, left 19 Schiotz. There is no sign of inflammation or further degeneration. The signs of retinitis striata, which were present before the operation, persisted after the operation.

The second case is a girl of twenty-six, with detachment of the inferior temporal quadrant of the retina, cause undetermined. Cystic degeneration of the retina was noted in the periphery. On April 5, 1932, under avertin anaesthesia the Guist procedure was employed with nineteen trephine openings. Viscous subretinal material escaped. Her vision is now 20/20 with no scotoma except for the peripheral field defect dependent upon the area which was destroyed by the cautery. The intraocular tension remained quite low for some time, but now has improved.

I feel that if these cases continue through a number of years as well as they have for a number of months, the procedure has much to recommend it. Complete rest and careful nursing care after the operation are very important.

Dr. Peter Kronfeld, Chicago: I am greatly impressed with Dr. McKeown's paper, but not by the high percentage of cures because I do not believe the results of operations for detachment of the retina can be expressed in figures. The figures show too much variation, although we have reason to believe the results are the same. At the last meeting of the German Ophthalmological Society in Leipzig, which I had the privilege of attending, Guist reported cures in as many as seventy-seven per cent of his uncomplicated cases. Dr. McKeown gives thirty-nine per cent, and Dr. Gradle fifteen per cent. We have every reason to believe that Guist, McKeown and Gradle are men of equal skill. The difference must lie somewhere else.

I am more impressed by the few complications that Dr. McKeown enumerated today. I have been doing the Guist operation for a year and a half (eight or nine cases) and I am still afraid of the dangers involved in this operation. Some complications arise from interference with the region of the ora serrata. In the first case Dr. McKeown presented today you remember he made three to five trephine holes in the immediate neighborhood of the ora serrata, parallel to the limbus. I would strongly advise you not to do such a thing. The ora serrata is a dangerous place because it is a region you cannot see with the ophthalmoscope. You have no idea how thick or how thin the choroid is in that region. Therefore I usually approach the ora serrata at two or three places only, and I do not go along the ora serrata as Dr. McKeown apparently does.

The other complication is interference with a large blood vessel. We cannot say in which direction the vorticose veins go after they enter the eye, and the vorticose sinuses vary a great deal in extent and location. I personally have seen quite a few serious hemorrhages during the operation.

The results I have had with the Guist method were very satisfactory, but I would not advise any beginner to try it. It may be all right in the hands of the experienced surgeon.

At this time I would like to mention that Lindner has apparently given up the Guist method. When I saw Lindner early in July of this year he was greatly impressed by the good results that Weve and others reported with diathermic micro-coagulation. The principle is the same — we are trying to surround the diseased portion of the retina by a row of artificial adhesions produced either by the potassium hydroxide stick, or, according to the new method, by electro-thermic coagulation, which in my opinion and that of others is a much simpler and safer method. Lindner is using the potassium hydroxide method (six per cent solution in water) for holes in the macula only; he undermines the choroid in those cases (choroidialysis) and then injects the KOH into the suprachoroidal space.

For those of you who are seeing only a few cases of retinal detachment and have not had much experience with the Guist method, I would recommend that you adopt the new method of diathermic micro-coagulation.

Dr. Hugh S. McKeown, closing: I want to thank the men who have discussed my paper. I have not much to add except detailed reports of our cases which are printed in the paper, showing the complications that arise, and in our conclusions we consider the postoperative care, which is important.

Dr. Gradle is correct, that with our limited experience it is too soon for us to give a definite opinion of this technique. The cases we have reported include both private cases and those done on Dr. Wheeler's service at the Medical Center, and the reason we are elated is that the results are so much better than with any other procedure.

Dr. Kronfeld spoke of the difference in percentages. The thirty-nine per cent of cures is based on all cases operated. I firmly believe that we can now increase that percentage of cures by refusing to operate on the apparently hopeless cases. Our group report includes cases from eight to seventy-one years of age, and detachments five days old to some that have been separated for two and a half years.

We intend continuation of this procedure as long as we are getting good results, and I hope to have a report later by which the ophthalmologist may be aided in determining what cases are suitable for operation.

MONOCULAR MYOPIA*

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SOME THREE HUNDRED years ago Kepler pointed out that an unusually large number of myopes are to be found among students and others engaged in work requiring close application of the eyes. Since that time opinion has been divided as to the interpretation of this finding. There is no doubt of the fact which has been amply confirmed by many investigators, but the question has not been settled whether eye strain causes myopia, whether eye strain causes an increase in myopia already present, or whether the occurrence of myopia predisposes the individual to professions and occupations in which close application of the eyes is an important element.

I shall not attempt here to review the voluminous literature on the subject, nor do I hope by this small contribution to settle a problem of such age-old contentiousness as this one. It will suffice to point out that no direct evidence has ever been advanced to prove that myopia may be produced or increased by eye strain. The most direct attempt at an unravelling of this problem in recent years is that of Sourasky,[1] who studied the frequency and degree of myopia among the Jewish boys and girls of London, comparing them with that of their English schoolmates. The Jewish boys chosen were ones who spent two hours daily in Hebrew school in addition to their regular grade school, and thus were subjected to considerably more eye strain than either their English schoolmates or their sisters. Sourasky found that there was, indeed, more myopia among the Jewish boys than among the non-Jewish boys, but there was a corresponding excess of myopia among the Jewish girls as compared with the non-Jewish girls. He was forced to conclude, therefore, that the differences found were racial, and not due to the extra hours of study.

* From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

Among the numerous aspects of this much debated subject, there is none more interesting than that dealing with the so-called "refraction curve," i.e., the frequency distribution of errors of refraction in the general population. It has been established by numerous observers that the distribution of errors of refraction in any large homogenous population exhibits certain well-defined characteristics. The most frequent state of refraction, that is, the peak of the curve, is close to +0.50 dioptres. From this peak the frequency falls off at first rapidly and then more slowly on either side. Up to + 5. and down to — 4. the two arms of the curve are practically symmetrical, but beyond — 4. there is to be found a considerable excess of myopes over the corresponding groups of hyperopes.

Those who believe that eye strain causes an increase in myopia explain this asymmetry of the refraction curve as resulting quite plausibly from the shifting of a number of individuals from their natural positions as low myopes to the category of high myopia as the result of eye strain. It is pointed out that the frequency curves of other bodily measurements, such as weight or height, are, in general, symmetrical. There are, for instance, approximately as many men in a given population who are one foot shorter than the average as there are one foot taller than the average. Recent analyses by Kronfeld[2] and Rosengren[3] have called attention to the weakness of this argument. It is obvious that the asymmetry of the curve is to be attributed to some factor other than the chance variation in the size of the eyeball, but there is nothing in the data so far given to prove that this additional factor is environmental or hereditary. One group of cases which has not previously been studied in relation to this problem is the group of cases showing myopia in one eye only. These cases present unique advantages for the study of the pathogenesis of myopia for in them the factor of excessive reading, while not completely absent, is, at least, reduced to a minimum. Many of them use the more normal eye for reading as well as for seeing in the distance. Those who read with the myopic eye have such a poor fusion faculty that they have no tendency to undue convergence, — the factor which above all has been held responsible for the alleged effect of reading in producing or increasing myopia.

For the purposes of this study we have selected from among 12,500 consecutive cases examined in private practice all cases of monocular myopia and marked myopic anisometropia, the selection being made according to the following rules:

1. When astigmatism was present the mean of the refractive error of the major and minor axes was taken as the refractive error.
2. Only cases showing a myopia of one diopter or more were included.
3. In myopic anisometropia only those cases were included in which the myopia of one eye was at least twice as great and at least one diopter more than that of the other eye.
4. All individuals of 60 years or more were excluded from the group.

In order to make these restrictions more clear, we present a list illustrating the *minimal* degree of myopia of one eye (A) for a given refractive error of the other eye (B) which would allow the case to be included in our selected group:

B 0 or +
A — 1.00

B — 0.50
A — 1.50

B — 1.00
A — 2.00

B — 2.00
A — 4.00

B — 3.00
A — 6.00

Out of 12,500 consecutive cases, 136 were found which filled these requirements, and in which the myopia was not due to keratoconus or to sclerosis of the lens nucleus.

In reviewing the records of these 136 cases, the first fact which attracts attention is the great frequency with which a history, and usually also some objective evidence, is obtained indicating severe inflammation of the myopic eye in childhood. In all, nineteen cases gave such evidence. The number of these cases is far too large to be merely a coincidence. This group

merits serious study and will be considered below. For the present, these cases are removed from the group of monocular myopes, and leave as a remainder only those cases in which an hereditary or congenital cause for the myopia exists, all accidental injuries and disease having been eliminated, and the effect of eye strain having been reduced to a minimum.

For purposes of comparison, a sample of 3,044 cases was selected at random from the same group of 12,500 cases. All instances of intraocular disease, all cases of keratoconus or corneal scars, all individuals sixty years or over were excluded. Astigmatism was treated in the same way as in the monocular myopes. In order to emphasize in this group the growth factors affecting the size of the eyeball, the average refraction of the two eyes was taken as the refraction of each case. The frequency distribution of refractive errors in this group is shown in Fig. 1 and Table I. It exhibits the usual characteristics. The highest frequency, the mode, occurs at + 0.50. The segments of the curve from + 1.00 to + 5.00, and from 0 to — 4.00 are symmetrical about a mean of + 0.503. Beyond these limits, there was the usual marked excess of myopes as compared with hyperopes.

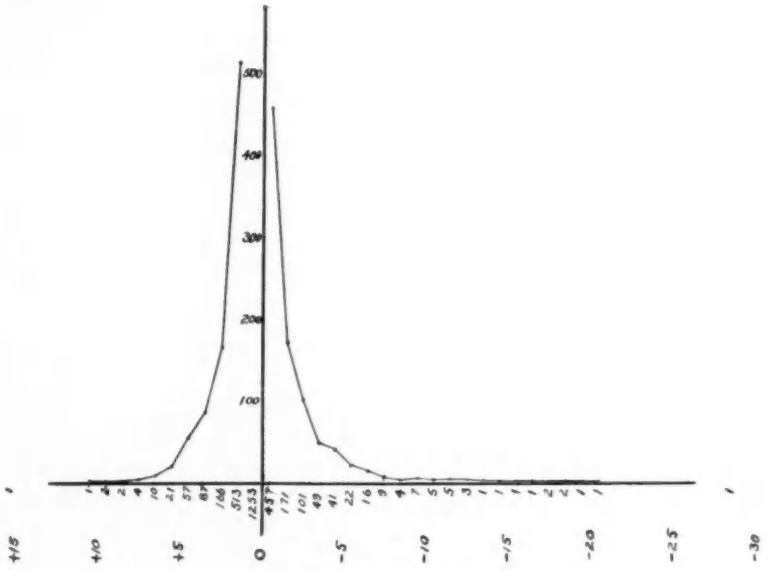

FIG. 1—Friedenwald)—Frequency distribution of refraction errors in 3,000 cases. The peak of the curve at + 0.50 dioptres is omitted.

TABLE I

Frequency of Various Degrees of Refraction among 3044 Cases

Refraction	Frequency	Refraction	Frequency
+ 15.12 to + 16.00	1	— 5.00 to — 5.87	22
—— ——	—	— 6.00 to — 6.87	16
—— ——	—	— 7.00 to — 7.87	9
+ 10.12 to + 11.00	1	— 8.00 to — 8.87	4
+ 9.12 to + 10.00	2	— 9.00 to — 9.87	7
+ 8.12 to + 9.00	2	— 10.00 to — 10.87	5
+ 7.12 to + 8.00	4	— 11.00 to — 11.87	5
+ 6.12 to + 7.00	10	— 12.00 to — 12.87	3
+ 5.12 to + 6.00	21	— 13.00 to — 13.87	1
+ 4.12 to + 5.00	57	— 14.00 to — 14.87	1
+ 3.12 to + 4.00	87	— 15.00 to — 15.87	1
+ 2.12 to + 3.00	166	— 16.00 to — 16.87	1
+ 1.12 to + 2.00	513	— 17.00 to — 17.87	2
+ 0.12 to + 1.00	1253	— 18.00 to — 18.87	2
0.0 to — 0.87	457	— 19.00 to — 19.87	1
— 1.00 to — 1.87	171	— 20.00 to — 20.87	1
— 2.00 to — 2.87	101	—— ——	—
— 3.00 to — 3.87	49	—— ——	—
— 4.00 to — 4.87	41	— 28.00 to — 28.87	1

The first problem to be attacked is a comparison between the frequency distribution of the degrees of myopia of the monocular myopes in the larger group. The data are shown in Fig. 2 where the two curves are reduced to the same proportions, and in Table II. A comparison of the two curves in Fig. 2 makes it immediately evident that there is an even greater relative excess of high myopes in the group of monocular myopes than in the group of binocular myopes. Is this difference significant, or is it due merely to the chance variations in the relatively small

TABLE II

Frequency of Various Degrees of Myopia

	Binocular Myopia			Monocular Myopia		
Refraction	Frequency	%	Excess over corresponding hyperopes	Frequency	%	Excess over corresponding hyperopes
— 1.00 to — 1.87	171	38.62	21	21	17.95	
— 2.00 to — 2.87	101	22.78		17	14.53	
— 3.00 to — 3.87	49	11.05	0	13	11.12	1
— 4.00 to — 4.87	41	9.24	16	9	7.69	4
— 5.00 to — 6.87	38	8.56	24	18	15.40	15
— 7.00 to — 8.87	13	2.93	9	12	10.27	11
— 9.00 to — 12.87	20	4.51	19	4	3.41	4
— 13.00 to — 16.87	4	0.90	3	7	5.97	7
— 17.00 to — 20.87	6	1.36	6	10	8.55	10
— 21.00 to — 24.87	0	0	0	3	2.56	3
— 25.00 to — 28.87	1	0.22	1	1	0.85	1
— 29.00 to — 32.87	0	0	0	1	0.86	1
— 33.00 to — 36.87	0	0	0	1	0.85	1

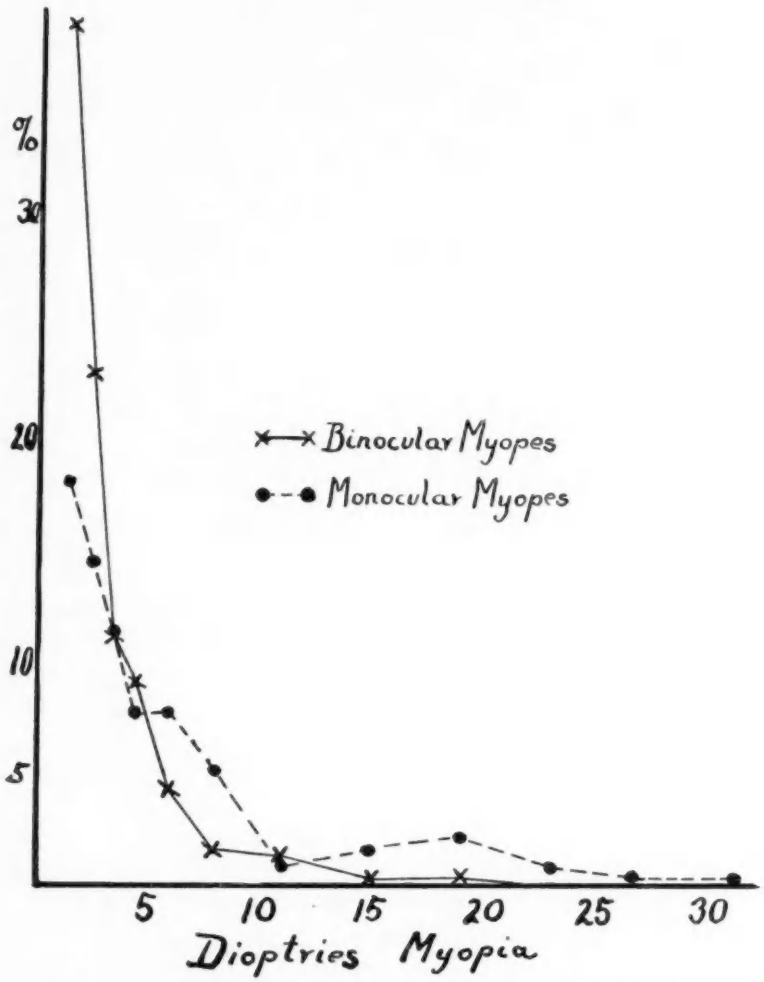

FIG. 2—(Friedenwald)—Percentage of frequency distribution of refraction errors among monocular and binocular myopes.

samples with which we are dealing? This question can be answered by the application of the so-called "Chi-square test," a statistical procedure which is designed for the solution of precisely this sort of problem.* The calculations required to per-

* This procedure is described in numerous text books of statistics, see, for instance, Raymond Pearl: Medical Biometry and Statistics, 2nd ed., Saunders Co., Philadelphia, 1930, pp. 315 and ff. The probability that the differences in the two sets of observations may result merely from random sampling is computed with the aid of "Tables of Statisticians and Biometricians" by Karl Pearson, Cambridge University Press, 1914.

TABLE III

Chi-Squared Test for the Significance of the Differences in Distribution of Degrees of Refraction Between Monocular and Binocular Myopes

REFRACTION	f	f_1	$f + f_1$	$\frac{f}{n}$	$\frac{f_1}{n_1}$	$\frac{f}{n} - \frac{f_1}{n_1}$	$\left[\frac{f}{n} - \frac{f_1}{n_1}\right]^2$	$\frac{\left[\frac{f}{n} - \frac{f_1}{n_1}\right]^2}{f + f_1}$
— 1.00 to — 1.87	171	21	192	.3852	.1795	.2057	.0423	.000210
— 2.00 to — 2.87	101	17	118	.2278	.1453	.0825	.0068	.00000577
— 3.00 to — 3.87	49	13	62	.1105	.1112	.0007	.0000	.000000008
— 4.00 to — 4.87	41	9	50	.0924	.0769	.0155	.0002	.0000048
— 5.00 to — 6.87	38	18	56	.0856	.1540	.0684	.0046	.0000836
— 7.00 to — 8.87	13	12	25	.0293	.1027	.0734	.0053	.000208
— 9.00 to —12.87	20	4	24	.0451	.0341	.0110	.000121	.00000504
—13.00 to —16.87	4	7	11	.0090	.0591	.0507	.0025	.000228
—17.00 to —20.87	6	10	16	.0136	.0855	.0719	.0051	.000323
—21.00 to —24.87	0	3	3	.0000	.0256	.0256	.0006	.000219
—25.00 to —28.87	1	1	2	.0022	.0085	.0063	.00003	.0000199
—29.00 to —32.87	0	1	1	.00000	.0086	.0086	.00007	.000074
—33.00 to —36.87	0	1	1	.0000	.0085	.0085	.00007	.000072
TOTAL	444	117		1.0007	1.0001			.001453

$$X^2 = n \cdot n_1 \sum \frac{\left(\frac{f}{n} - \frac{f_1}{n_1}\right)^2}{f + f_1} = 444 \times 117 \times .001453 = 75.5$$

From Pearson's Tables P for this value of X^2 is .000000

form this test are shown in Table III. The results of this test prove that the chance that two such distributions as we have found would result from the effects of random sampling is less than one in a million. The difference is therefore significant.

It is possible, however, that this significant difference might result from some other disparity between the two groups such as age, sex, or race, rather than from the characteristic differences of the two groups of cases which we were investigating. Simple tests showed that there was no significant difference in the sex or race of the patients, and the Chi-square test applied to their age distribution likewise revealed no significant difference. It may be concluded, therefore, that the difference found is an essential characteristic of the two groups analyzed, and is not to be attributed to the indirect influence of some adventitious factor which might accidentally bias the selection. It is perhaps of interest to note that a significant difference in age distribution was found in comparing each of the myopic groups with the general population from which they were chosen. Both groups of myopes showed an excess of cases in the second decade of life, a finding which is not surprising in view of the obvious fact that this is the age period when myopes as a rule first consult ophthalmologists. The discovery of this difference is an index of the delicacy of the statistical method used.

Turning our attention now to the curves of Fig. 2, the differences between which we now know are significant, we find that the most characteristic feature differentiating the two curves is the fact that whereas the frequency of myopia decreases regularly as one approaches the higher degrees in the group of binocular myopes, the frequency curve of the monocular myopes on the contrary shows a secondary hump which has its peak at about — 17. dioptres, and decreases more or less symmetrically on either side of this peak. Furthermore, the remainder of this curve, after deducting the cases corresponding to this hump evidently approaches a form symmetrical with the hyperopic arm of the general refraction curve. We may conclude, therefore, that the group of monocular myopes consists of two types of cases: first, a group the distribution of which is symmetrical with the hyperopic arm of the general refraction curve, and which, therefore, taken together with the hyperopes represent refraction errors attributable to normal variations in the length

of the eyeball. This group it seems reasonable to call *normal myopia.* Secondly, we have group of cases whose average refraction falls in the region of high myopia. A very large proportion of these cases show serious fundus changes, and have defective vision. It seems reasonable to call this group *pathological myopia.*

As to the cause of pathological myopia, we cannot at present draw any positive conclusions. On the negative side, we can, however, say that it is not due to inflammatory diseases of the eye in childhood, since these were excluded by our original selection of the cases. Similarly it cannot be due to excessive reading since we have reduced this factor to a minimum in our cases. It must, therefore, be due to some, as yet unknown, congenital, and, no doubt, hereditary weakness of the coats of the eyeball.

If our conclusions, thus far, are correct, it is reasonable to expect a similar group of pathological myopes among the binocular myopes, and if, as appears from the data so far, only normal myopia and pathological myopia enter into the consideration, then we can discover the frequency distribution of the patholog-

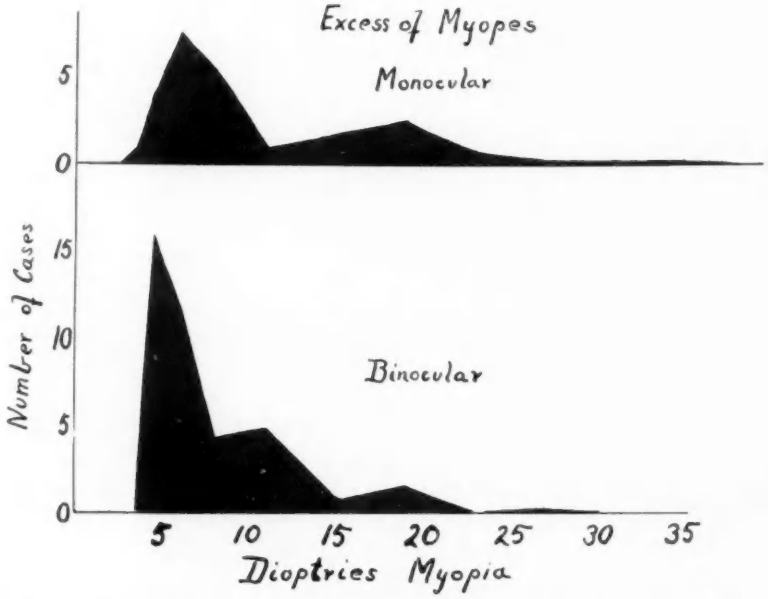

FIG. 3—(Friedenwald)—Frequency distribution of the excess of myopes over the numbers required for symmetry with the hyperopic arm the general refraction curve.

ical myopes by the simple procedure of subtracting from each frequency group among both the monocular and binocular myopes the number of cases required to leave the curve symmetrical with the hyperopic arm of the general refraction curve. The results of this calculation are shown in the fourth and seventh columns of Table II, and in Fig. 3.

Owing to the small number of cases falling under this heading, numerous irregularities and differences are found between the two curves in Fig. 3. The chief of these consists in the fact that the pathological myopes among the binocular myopes show relatively greater frequencies in the range of medium myopia (—4 to —9) while those found among the monocular myopes show relatively greater frequencies in the higher degrees of myopia. The difference between the frequencies in the two series, however, is only 10 per cent, and the probable error of the difference as shown in Table IV is 7.8 per cent or 8.3 per cent, depending on the mode of calculation. Such a small excess over the probable error is not generally considered significant. It must be noted, also, that the figures for the "pathological myopia" in the lower degrees of refractive error represent only a small fraction of the myopes in these groups, and that, therefore, small irregularities in the original curves attributable to the errors of sampling, if they affected either the myopes of this degree or the corresponding hyperopes would cause very large

TABLE IV

Pathological Myopia

	Monocular		Binocular		Difference	Total	
	No. of cases	%	No. of cases	%	%	No. of cases	%
Under 9 dioptres	31	54(p_1)	49	64(p_2)	10	80	59(p_0)
Over 9 dioptres	27	46(q_1)	28	36(q_2)	10	55	41(q_0)
Total	58(n_1)		77(n_2)			135($n_1 + n_2$)	

The probable error of the difference may be calculated according to either of the following two formulas:

$$(1) \qquad E_{1.2} = \left[p_0 q_0 \left(\frac{1}{n_1} + \frac{1}{n_2}\right)\right]^{\frac{1}{2}} = \left[0.59 \times 0.41 \left(\frac{1}{58} + \frac{1}{77}\right)\right]^{\frac{1}{2}} = 7.8\%$$

or

$$(2) \qquad E_{1.2} = \left[\frac{p_1 q_1}{n_1} + \frac{p_2 q_2}{n_2}\right]^{\frac{1}{2}} = \left[\frac{0.54 \times 0.46}{58} + \frac{0.64 \times 0.36}{77}\right]^{\frac{1}{2}} = 8.3\%$$

percentage errors in calculated numbers of pathological myopes of low degrees of myopia. We can conclude, therefore, that in all probability no such phenomenon as reading myopia exists, that if it does exist at all it is manifested only in the moderate degrees of myopia, and certainly has nothing to do with high myopia.

We must return now to a consideration of those cases of monocular myopia in which evidence was found of juvenile inflammation of the affected eye.

It has, of course, long been known that children suffering from phlyctenular keratitis, interstitial keratitis, and even from extensive chorioretinitis frequently become myopic, but this has been interpreted as the result of the excessive effort which persons with such poor vision have to make in order to see, and the group of these cases had been reckoned with the so-called reading myopias. The cases in our series are presented in Table V. The number of these cases occurring among monocular myopes is so large as to rule out entirely any notion of chance coincidence, and is sufficient to prove that these ocular troubles in themselves, and without any associated eye strain, can and do produce myopia of even very high grades. It is interesting to speculate about the possible cause of myopia in these cases. The types of injuries and diseases represented by this group are quite varied, and we may add to them the cases of myopia following juvenile hemorrhages in the vitreous previously reported by Dr. Harry Friedenwald,[4] and not included in this series. It is difficult to find a common denominator among these varied conditions. In the affections of the anterior segment of the eyeball included within this list, blepharospasm is a dominant feature, but in the cases of choroiditis, vitreous hemorrhages, and optic atrophy this symptom is absent. It would seem that the greatest common denominator among all these inflammatory troubles is the congestion of the eye ball, and this would appear to be the most likely factor responsible for myopia in these cases. Long-standing and persistent congestion of the eyeball may, therefore, be suspected to be injurious to the sclera, and predisposing to the development of myopia.

TABLE V — *Secondary Myopia*

Case	Age		Refraction	Vision	Findings	Cause
1.	30	R.E.	— 20.00	Poor light projection	Post neuritic optic atrophy	Unknown, occurred in childhood
		L.E.	+ 0.50 — 0.37 × 15	20/15	Normal	
2.	45	L.E.	— 13.00 — 3.00 × 105	20/80	Corneal scar	Phlyctenular keratitis
		R.E.	— 2.50 × 100	20/40	Normal	
3.	30	L.E.	— 13.00	Fingers 1 ft.	Corneal scar	Injury in childhood
		R.E.	+ 0.25	20/15	Normal	
4.	9	R.E.	— 13.00	20/40	Corneal scar	Phlyctenular keratitis
		L.E.	— 6.00	20/40	Corneal scar	Phlyctenular keratitis
5.	46	L.E.	— 9.00 — 4.00 × 90	20/200	Corneal scar	Followed measles
		R.E.	+ 0.50 × 15	20/20	Normal	
6.	38	R.E.	— 7.00 — 2.00 × 135	20/120	Corneal scar	Phlyctenular keratitis
		L.E.	— 4.00	20/60	Corneal scar	Phlyctenular keratitis
7.	36	L.E.	— 7.00	20/80	Corneal scar	Phlyctenular keratitis
		R.E.	+ 0.25	20/20	Normal	
8.	47	R.E.	— 6.00	20/200	Corneal scar	Phlyctenular keratitis
		L.E.	+ 1.25 — 1.75 × 180	20/48	Slight corneal scar, later detachment of retina following injury	Phlyctenular keratitis
9.	55	L.E.	— 6.00	20/200	Corneal scar	Occurred in childhood
		R.E.	— 3.00	20/80	Corneal scar	Occurred in childhood

Case	Age		Refraction	Vision	Findings	Cause
10.	29	R.E.	$-4.00 \; -4.50 \times 175$	20/60	Choroidal scars	Exudative choroiditis
		L.E.	$+0.75 \; -2.75 \times 180$	20/48	Normal	
11.	42	R.E.	$-4.00 \; -4.00 \times 180$	20/200	Corneal scar	Cut with stick at age of 13 years
		L.E.	$-0.50 \; -1.50 \times 75$	20/30	Normal	
12.	52	L.E.	$-3.50 \; -2.50 \times 135$	20/60	Corneal scars	Occurred in childhood
		R.E.	$-1.00 \; -2.00 \times 180$	20/60	Corneal scars	Occurred in childhood
13.	23	R.E.	-3.00	20/80	Corneal scar; iridectomy	Cut with tin can at age of 12 yrs.
		L.E.	$+0.25 \times 180$	20/20	Normal	
14.	54	R.E.	-3.00	20/30	Corneal scar	Phlyctenular keratitis
		L.E.	$+3.00 \times 115$	20/24	Normal	
15.	24	R.E.	$-2.00 \; -1.00 \times 165$	20/80	Scar of penetrating wound	Cut by explosion at age of 4 yrs.
		L.E.	-0.25	20/20	Normal	
16.	21	L.E.	$-1.25 \; -3.00 \times 165$	20/48	Corneal scars	Phlyctenular keratitis
		R.E.	$+2.00 \times 105$	20/30	Corneal scars	Phlyctenular keratitis
17.	35	L.E.	$-1.00 \; -3.00 \times 165$	20/80	Corneal scars	Occurred in childhood
		R.E.	$+2.00 \; -1.00 \times 180$	20/20	Normal	
18.	28	R.E.	$-0.50 \; -2.50 \times 165$	20/60	Corneal scar	Struck with a buckle at age of 6 years.
		L.E.	$+2.50 \times 110$	20/38	Amblyopic divergent strabismus	Automobile accident with fracture of skull at age of 16 years
19.	27	L.E.	-3.00×180	20/200	Corneal scars	Phlyctenular keratitis
		R.E.	$+0.50$	20/20	Normal	

SUMMARY

The foregoing analysis of 136 cases of monocular myopia and marked myopic anisometropia reveals the fact that these cases may be divided into three groups:

(1) Normal myopia 43%
(2) Pathological myopia 42%
(3) Secondary myopia 14%

If one excludes secondary myopia from the group, about half of the remaining primary myopes among the monocular myopes can be considered as resulting from normal variations in the size of the eyeball, while the other half are to be attributed to an intrinsic weakness of the coats of the globe. Among the primary binocular myopes, pathological myopia is relatively less common, amounting to 17.3 per cent of this group. The absolute number of binocular pathological myopes is, however, greater than that of monocular pathological myopes, in a ratio which may be estimated as three or four to one.

The procedure which we have outlined enables one to isolate a group of myopes whose frequency distribution is symmetrical with that of the hyperopic arm of the general refraction curve, and which, in accord with other writers on this subject, we have considered as due to normal variations in size of the eyeball. It must not be thought, however, that such a simple statement is completely satisfactory. Many writers have shown that even when the asymmetry of the refraction curve has been eliminated, the frequency which remains does not exactly fit the normal frequency curve which is adequate to fit other groups of observations or random variations. According to Kronfeld, the refraction curve corrected for symmetry shows an excess at the peak, and, also, an excess in the region of high refractive errors as compared with the normal frequency curve. This is perhaps not so very surprising. The total refraction of the eye is dependent in the first instance upon the length of the globe, but is, also, affected by the curvature of the cornea, and the position and curvatures of the lens. At the present time, we do not know how these various factors vary nor in what way they are correlated with one another.

The group of pathological myopes which we have isolated from the total myopia population coincides with the group of

"conus myopia" described by Scheerer[5]; that is, myopes showing signs of stretching of the choroid. Scheerer's data, however, were not adequate to determine whether or not conus myopia could be attributed to the effects of eye strain. The findings in our group of cases: (a) that pathological myopia is relatively more frequent among monocular myopes than among binocular myopes, and (b) that the frequency distribution of various errors of refraction in these two groups of pathological myopes is essentially the same, suffices to show that pathological myopia is not the result of, nor affected by, eye strain. These results raise grave doubts as to whether there exist any cases at all which may properly be called "reading myopia."

Secondary myopia, also, occurs relatively more frequently among monocular myopes than among binocular myopes, and since the vision of the unaffected eye is in the majority of these cases normal, the development of the myopia in these cases cannot be attributed to eye strain. The only common factor which could be found among the medley of cases falling in this group is that of intraocular congestion, and it seems likely, therefore, that long-standing congestion of the eyeball in infancy or childhood may weaken the sclera. If this conclusion is correct it furnishes us with a rational basis for the understanding of the remarkable successes which Dr. Wiener[6] has reported with the long-continued use of adrenalin. Unfortunately the absence of an adequate control series makes it impossible to evaluate the significance of the results which Dr. Wiener has reported.

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DISCUSSION

Dr. Meyer Wiener, St. Louis: The author, in his admirable essay on uniocular myopia, refers to the preponderance of high myopes over high hyperopes. It seems to me that this can be explained, in that, while all eyes are hyperopic in the beginning, the hyperopia never increases, while the tendency of the eye to elongate is ever present. His belief that there is some factor other than reading, or close application of the eyes

creating the excess of myopes only confirms the careful and laborious work of Steiger and other investigators of this subject. The pathological myopias, unilaterial as well as bilateral, are almost always congenital, or at least are observed in the first few years of life. While a great number of them become progressively worse, I have watched many over a period of years, with no change in either fundus appearance or refraction. I do not believe that different grades of either hyperopia or myopia can be separated.

Donders' measurements seemed to prove that the difference in refraction did not lie in corneal changes, but in the axial length.

In a very large proportion of cases hereditary predisposition unquestionably exists. We must not confound the terms "hereditary" and "congenital" under the former. We must consider the future of the eye as the tendency to develop to that which it was potentially ordained at the time of conception. This is not necessarily a fixed thing, but can be altered by various factors. We do know that if we take two bowls of tadpoles of the same age and feed one with ordinary food, giving the other the same with the addition of pituitary extract, the second lot will attain a much larger growth, even though both groups come from the same hatching. Therefore it is fair to assume that the hereditary tendency to stretching of the sclera, or progressive myopia, may be altered or averted under proper conditions. Some authorities have endeavored to compare animal myopia with the human myopia. This is not comparable, for while all babies, with very few exceptions, are born hyperopes, showing increase in refraction with growth, animals show the same refraction when full grown as that found in the young.

In studying a group of forty-one cases of uniocular myopia observed by me in the last few years, eleven showed an increase in the refraction of one or both eyes over a period of from one to twelve years, varying from 0.5 diopters to as much as 4.0 diopters. Four of them increased so that the myopia was binocular at the last observation. One would naturally assume that a large proportion of those cases of binocular myopia in which the degree is different in the two eyes had been either hyperopic in both eyes or hyperopic in one eye and myopic in the other, at an earlier period. I believe that a study of the same difference in hyperopes, especially those which have shown a decrease in the amount of plus, should be made for comparison or control. As an illustration of my point let me briefly outline the history of one case.

Miss M. K., at the age of four, was seen by me ten years ago, with a total refraction of +6.0 diopters in the right eye, and +9.0 diopters in the left eye. The left eye converged about 30°, first noticed by the mother a year before. The vision with correction was 15/12 in the right, and 15/300 in the left eye. The eyes became less hyperopic. On November 9, 1929, she had a total of +1.25 diopters in the right eye, and +3.50 in the left. January 25, 1930, she had +0.5 diopters in the right eye, and +3.0 diopters in the left. She was placed on epinephrine drops 1-1000 instilled in the eyes three times a day, but the refraction continued to increase. On April 28, 1932, refraction right eye —1.0 diopters, —0.5 D.

cyl., axis 0. Left, +2.5 diopters. She was then sent to an internist for a thorough workup, with especial attention to her endocrine function. The report showed B.M.R. —14, sugar tolerance slightly increased; blood, Kahn negative; stereo of skull negative; Goetsch test, reaction to adrenalin moderately diminished, suggesting hypoadrenalism; blood calcium 11.2 mmgr. (normal). Dry skin and hair, plus the examination, suggest hypothyroidism. Impression: Hypothyroidism, hypoadrenalism. She has been taking small doses of thyroid extract, but at the time of the last examination, September 8, 1932, her refraction had not changed.

I do not feel that the statement of the author that "the significance of my observations on the use of adrenalin cannot be properly evaluated on account of lack of control cases" is altogether correct. I have not included in my tables any case of myopia where the patient had not shown definite evidence of actively increasing refraction. Dr. Gradle in his discussion gave thirty cases treated with adrenalin, and thirty cases in which it was not used. The results were striking, when compared. In the present series of 41 cases, for example, only eleven showed a tendency to progress. The other thirty were not placed on adrenalin. I submit a short history of one of these eleven cases.

T. H., was first seen by me on February 1, 1926, at the age of nine. Vision, right with —0.5 D. cyl., axis 15 = 15/12; left, +0.5 d. cyl., axis 90 = 15/12. Four years later his refraction in the right was S. —0.5 D., —0.5 D. cyl., axis 0; left, S. —1.0 d., —0.75 D. cyl., axis 0. An increase of 0.5 D. in the right and 1.75 D. in the left.

He was then placed on adrenalin and urged to take a lot of exercise. His refraction has not changed in the last two years. His mother and his sister are high myopes.

Why the refraction increases in one eye, sometimes the more myopic, at other times the hyperopic, is inexplainable. Why should a person with tuberculosis, syphilis, or tonsil infection develop iritis in one eye only?

S. G., age 15, revealed a refractive error of S. —16.0 D. in the right eye, and S. +1.5 D. in the left. Five years later the refraction was S. —20.0 in the right, and still S. +1.5 D. in the left.

It seems that the uniocular myopias of extremely high degree do not have the same tendency to progress as those of moderate or slight degree. This is also true of the binocular form. I am inclined to believe that monocular myopia cannot be considered as a separate entity from the binocular variety.

Dr. E. M. Josephson, New York: Dr. Friedenwald's mention of vascular changes incident to the pathology of myopia, and Dr. Wiener's similar data impels me to report my own observations on a series of cases of monocular myopia.

In checking over the work of Baillart on the retinal circulation I observed with particular frequency in the cases of monocular myopia that it was possible by comparatively slight pressure on the myopic eye to cause the appearance of the arterial pulse and then obliteration of the vessel, before any change could be affected in the veins. Apparently the

venous pressure was higher than the arterial, a state of affairs that could hardly be rational. No doubt the phenomenon is a paradox, due to difference in structure of the vessels and difference of response to stretching of the eyeball by the disease process. It is noteworthy that the amount of pressure required to secure the obliteration of the arterial lumen was very slight, hardly greater than the pressure exerted by the extrinsic muscles of the eye. This observation might serve to throw some light on the progressive nature of myopia. Incidentally, the tension in these cases was within the range of normal.

I submit the observation for what it is worth and ask Dr. Friedenwald whether his observations enable him to throw any additional light on the matter.

DR. JONAS S. FRIEDENWALD, closing: I am glad that Dr. Wiener has emphasized the distinction between hereditary and congenital. If we believe that progressive myopia results from an inherited tendency, we are not forced to conclude that neither the ordinary factors of environment, such as school work, nor any unusual factors introduced by way of treatment are necessarily without effect. All that one may conclude from the present analysis is that the asymmetry of the refraction curve does not result from the effects of reading on potentially myopic eyes.

Dr. Wiener agrees that there is no significant difference between monocular and binocular myopia. This is the fundamental hypothesis upon which this work was based. So far as the essential cause of pathological or progressive myopia is concerned, it seems to me more satisfactory to assume an inherited weakness of the sclera rather than to double the mystery by assuming an unknown relation between the eyes and the endocrine system. This would certainly be a more satisfactory explanation of the pathogensis of monocular myopia than to subsume this problem under the general mystery of the selective localization of infectious diseases, as Dr. Wiener suggests. It is interesting that the analysis of monocular myopia secondary to ocular injury or inflammation in childhood provides a rationale for the treatment of myopia by the Wiener method without necessitating the assumption of distant endocrine effects.

So far as the evaluation of the efficiency of the adrenalin therapy is concerned, I feel we are still much in the dark. We are all familiar with the fact that cases of progressive myopia do not, as a general rule, progress with steady uniformity. Very frequently we find that they progress rapidly for a period of months or years and then remain stationary either for a limited period or permanently. What we require to know is whether these stationary periods are longer or more frequent in treated cases than in untreated cases, whether the rate of progress during the periods of progression is more or less great. Our present study, however, is only very indirectly related to this question.

I have had no experience with the problem of the pulsation of the retinal vessels in relation to the pathogenesis of myopia.

COMPLETE DISCISSION OF THE CRYSTALLINE LENS

*Preliminary Report of Clinical and Experimental Studies**

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CONGENITAL CATARACT caused blindness in approximately fifteen per cent of the pupils in residential and day schools for the blind in the United States in 1931. This condition accounted for more blindness than any other single disease (Table I). In one New York school for the blind[1] twenty-seven children (twenty per cent) were blind because of congenital cataract, and operation on thirteen of these patients (ten per cent of the inmates) was unsuccessful. Thus the selection of the operation for congenital and juvenile cataract is important in the prevention of blindness.

In this consideration of the operation known as "complete discission of the crystalline lens" the term "discission" is defined and limited in accordance with its derivation, to a cutting through of the entire lens with its capsule, hence, it is usually referred to as the "through-and-through" operation.

Complete discission of the lens has been employed successfully in treating congenital, juvenile, and traumatic cataracts and is also useful in myopia of high degree.

The reasons for presenting the subject of complete discission of the crystalline lens may be summarized as follows:

1. The high percentage of blindness due to congenital cataract and the large number of unsuccessful operations for this condition.

2. Complete discission of the lens has been criticized by eminent surgeons, but our experience in the majority of cases apparently does not support their objections.

* From the Lighthouse Clinic for the Prevention of Blindness. Research funds from the Ophthalmological Foundation, Inc.

*Diseases of the Eye Causing Blindness Among Pupils in Residential and Day Schools for the Blind in the United States in 1930-1931**

	Total	Per Cent
Number of Pupils	5,286	100.
DISEASE		
Congenital Cataract	782	14.8
Other Congenital Causes	734	13.9
Ophthalmia Neonatorum	717	13.6
Trachoma	69	1.3
Progressive Myopia	142	2.7
Interstitial Keratitis	161	3.0
Phlyctenular Keratitis	45	.9
Optic Nerve Atrophy	655	12.4
Chorioretinitis	155	2.9
Retinitis Pigmentosa	97	1.8
Accidents	423	8.0
Infantile Glaucoma	153	2.9
Other Causes	1,153	21.8

3. The technic herein described differs from Ziegler's[2] original technic in that a subconjunctival incision at the limbus has been substituted for the corneal incision, and the single straight incision through the lens has apparently yielded as good results as the inverted V-shaped incision and is a simpler procedure.

4. Preliminary iridectomy which was formerly combined with complete discission has not been performed in cases operated on in recent years. Possibly the results have been less successful in certain cases because preliminary iridectomy has not been performed.

5. This operation in our hands has afforded good results, comparable with the results of the older method of anterior capsulotomy. Postoperative reaction is apparently less in young patients. The number of operations is usually decreased. In the last five years, since the incision in the anterior and posterior capsules has been made longer and the single straight incision through the lens has entirely replaced Ziegler's inverted V-shaped incision, the results have been satisfactory except in older patients.

* Table prepared from data furnished by the National Society for the Prevention of Blindness.

History of Discission

In 1808, Percival Pott[3] discovered that a cataract would be "dissolved" by the action of the aqueous if the capsule of the lens was freely lacerated. In 1803, Hey[4] stated he always endeavored to tear off a portion of the capsule in order to let "the aqueous humor flow upon the broken cataract." To Saunders[5] (1811) must be accorded the honor of discovering and adapting the principle of discission of the lens in congenital cataract. He used the original model of Hey's knife-needle (sharpened on both edges). In 1830, Guthrie[6] completely divided the lens by an incision posterior to the iris using a curved knife devised by Langenbeck[7] which closely resembles the Ziegler knife-needle. No mention of complete discission of the lens by an incision anterior to the iris apparently appears in the literature prior to Ziegler's[8 9 10] publication of his practical method. Ziegler's operation was approved by Chance[11] in 1923 and Zentmayer[12] in 1922 found it useful in traumatic cataracts.

Preliminary iridectomy was practiced by von Graefe and was again proposed in 1871 by Meyer and de Montméja[13] who advised preliminary iridectomy in all patients over fifteen years of age. These surgeons performed iridectomy with anterior capsulotomy. More recently, Bell[14] suggested combining iridectomy with Ziegler's complete discission of the lens, and reported one hundred cases operated on by this method.

Although we saw no complications in the series of cases operated on by Bell, we agree with Radcliffe[15] that preliminary iridectomy, which was so valuable in the older methods of discission, is seldom necessary in complete discission of the lens in young patients. However, experience recently gained may indicate the advisability of performing preliminary iridectomy in patients over ten years of age if complete discission is to be performed in preference to linear extraction following anterior capsulotomy.

Advantages of Complete Discission

The advantages of complete discission of the lens appear to be:[16] Fewer operations are required to obtain the same results; less reaction is usually noted than after anterior capsulotomy; secondary increase in tension is less frequently encountered;

danger of infection is probably less, for the number of operative procedures is usually reduced; preliminary iridectomy seems to be unnecessary in young patients and certainly is not so important as in anterior capsulotomy.

Objections to Complete Discission

While the merits of complete discission are recognized by several ophthalmologists, adverse criticism has also been advanced.

Wilder[17] objects to the procedure on the basis of (1) undue reaction; (2) luxation of the lens by tearing the zonule; (3) delayed absorption of the cataract; (4) greater likelihood of the formation of membranous after-cataract.

Greenwood[18] and McReynolds[19] agree with Wilder. McReynolds adds that cutting into the vitreous causes slow absorption.

Fisher[20] prefers the Graefe incision, capsulotomy and peripheral iridectomy.

Wiener[21] who has had twenty-three years' experience with the Ziegler operation approves of this procedure, but objects to the subconjunctival incision at the limbus. He believes that Ziegler's method of making the puncture within the cornea should be followed.

Indications and Contraindications for Complete Discission

Age—In soft cataract complete discission of the lens may be performed from birth to the age of thirty-five years. The earlier an operation is performed the better, but the teething period should be avoided and we usually prefer to wait until the child is a year and a half to two years of age. Kirby[22] states that children with cataract develop amblyopia if they are not operated upon before four years of age. Elschnig[23] advocates operation as early as the first weeks of life in order to prevent amblyopia. It may be practical to perform this operation on older patients but severe reactions and high tension resulted in two patients between seventeen and twenty years of age. Discission of the anterior capsule was performed by Post[24] successfully on two patients, thirty-five and thirty-seven years of age, and Czermak has performed anterior capsulotomy in selected cases up to forty years of age. The age limit should also be considered in operating on the transparent lens in high myopia.

Obviously, the method is not suitable for all cases of congenital or juvenile cataract. For example, it should not be used for congenitally subluxated cataractous lenses, for the loosely-attached lens would recede from the instrument or might be completely luxated.

Phaco-anaphylaxis, unless desensitization is complete, may be a contraindication. Chronic infections which cannot be eliminated would make it advisable to perform anterior capsulotomy and linear extraction.

The slitlamp examination will show the relative size of the embryonic, fetal and adult nuclei. A diffuse grayish haze or brownish color, such as one frequently sees in senile lenses, indicates a hard sclerosed nucleus tending to resist absorption.

Preliminary Preparation for Operation

Determine the patient's sensitiveness to lens protein by means of the intracutaneous test with solutions of lens protein 1:10, 1:100, 1:1000. If he is hypersensitive he should be desensitized before operation by the method suggested by Verhoeff and Lemoine.[25] Several patients who have had congested eyeballs following operations were considered to have had phacogenetic, not phaco-allergic, endophthalmitis as intradermal tests with lens antigen were negative. One patient who had a zonular cataract[26] (Table III, Case 2) was sensitive to lens antigen. He was desensitized prior to operation and a good result was obtained.

The general condition of the patient should be determined. The Wassermann test should be made. The possibility of focal infections from the nasal accessory sinuses, teeth, tonsils, adenoids and other sources should receive careful consideration.

Examination of the eye should be made with especial attention to chronic obstruction and inflammation of the nasolacrimal passages.

Cultures from the conjunctival sac, on blood agar, should be made two or three days before operation. If pathogenic organisms are found, operation should be deferred.

Instruments Required

Speculum
One fixation forceps (with catch)

One fixation forceps (without catch)
One mousetooth forceps
Ziegler knife-needle with long curved blade (size selected according to diameter of the lens and pupil when dilated)
Spatula probe
Lid retractor

Preparation for the Operation

The pupil is completely dilated with atropine fortified by epinephrine. This method was advocated by Dascalopoulos[27] in operations for traumatic cataract. The advantages of subconjunctival epinephrine injections have been enumerated by Barkan.[28]

Technic of the Operation

After complete mydriasis is obtained the conjunctiva is nicked with scissors 6 millimeters from the limbus in an area dried and painted with a five per cent solution of mercurochrome. A well-curved Ziegler knife-needle is then passed subconjunctivally. The flat of the needle is held parallel to the plane of the iris and thrust through the sclera 0.5 millimeter from the limbus. The lens is cut through from below upward with a gentle sawing motion and a single straight incision is made. This incision has also been advocated by Parker. Our experience differs from that of Ziegler in two respects: First, Ziegler believes that it is never necessary to cut the secondary membrane, while four of our patients required secondary operations; second, that the corneal route is to be preferred to the subconjunctival method of approach because a better fulcrum is made and there is less danger of puncturing the iris or the ciliary body. Our belief is that the subconjunctival incision is safer from the standpoint of preventing infection. The corneal incision depends entirely upon its lymph supply for nourishment and is therefore more susceptible to infection.

Complications to Be Avoided

The lens should not be dislocated. This complication is usually avoided if a gentle sawing motion is used.

The iris should not be punctured and the edge of the pupil should not be nicked.

TABLE II — *Experimental Discission of the Lens*

	COMPLETE DISCISSION				ANTERIOR CAPSULOTOMY				REMARKS
	RABBITS		GUINEA PIGS		RABBITS		GUINEA PIGS		
	No. of Animals	No. of Eyes	No. of Animals	No. of Eyes	No. of Animals	No. of Eyes	No. of Animals	No. of Eyes	
Total Number of Animals and Eyes Operated On	26	29	2	4	25	30	2	4	
Complications:									
1. Glaucoma		3				2			
2. Hypotonia		4				4			
3. Panophthalmitis		1		1					
Subluxation of Lens		7				3			Occurred mostly during secondary operations
Results and Condition of Lens:									Operations were repeated on 21 eyes
Complete Absorption		9		3					
Partial Absorption		15							
Cataract Without Absorption		8		1		9			
Scar in the Anterior Capsule of the Lens						21		4	

When making the lens puncture below, care should be taken not to injure the ciliary body.

The danger of increased tension as a result of complete discission has been thoroughly investigated by Alvis.[29] His experiments with rabbits showed that simple discission of the anterior capsule and cortex were followed by plus tension and slow absorption, while complete discissions were followed by minus tension and rapid absorption.

Discussion of Our Experiments on Animal Eyes Tabulated in Table II

Twenty-five rabbits and four guinea-pigs were operated upon and observed from December, 1931, to June, 1932. The operations attempted were complete discission of the lens by a straight incision on one eye, and anterior capsulotomy by a single straight incision on the other eye. One hour before operating the pupils were dilated with atropine, one per cent, and epinephrine 1:1000. The eye was anesthetized with two per cent holocaine and four per cent cocaine before the operation. Tension was taken with the Bailliart corneal tonometer before the operation and twice weekly during the period of observation. A subconjunctival incision at the limbus with a Ziegler knife-needle was made in all but three rabbits; in the latter a corneal incision was made.

Increase of tension lasting several days was observed in five eyes; in one eye, on which an incomplete discission had been performed, the high tension persisted. On two of these eyes an incomplete discission and on the other three eyes a complete discission had been performed. A decrease of tension with marked reaction lasting two to five weeks developed in eight eyes, the distribution between the two types of operation being about equal. One rabbit and one guinea-pig developed panophthalmitis in one eye (in both complete discission of the lens had been performed). The ease with which the rabbits' lenses were subluxated in ten eyes (nine rabbits), especially during the secondary operations, was noteworthy. The guinea-pigs' eyes were so small and dark that the field of operation was obscured.

Complete discission of the lens was performed on thirty-three eyes (twenty-six rabbits and two guinea-pigs), including repeated operations. In nine of these, complete absorption of the lens was observed; in fifteen eyes there was partial absorption

of the soft lens matter. These animals are still under observation. Anterior capsulotomy was performed on thirty-four eyes. Rapid healing of the wound with scar formation in the anterior capsule or in the anterior layers of the lens invariably followed. On twenty-one of these eyes (fourteen rabbits) a secondary operation was necessary in order to produce swelling of the lens. Cataracts without absorption developed in twelve rabbits (seventeen eyes) equally distributed between the two types of operation. No secondary operation was undertaken on this group of animals because in certain of the animals the lens was subluxated and in the others the eyes were inflamed.

Results of Complete Discission of the Human Lens

Congenital Cataract

Of nineteen patients with congenital cataract upon whom complete discission of the lens was performed, six had postoperative complications. In one of these patients incarceration of the iris in the wound produced a slightly irregular pupil. Increased tension was noticed in four cases (Table III, 6, 18, 19, 21). In two cases (6, 18) linear extraction was necessary.

The patient in Case 18 obtained good vision with correction. In Case 21 the high tension subsided under miotics and the patient obtained good vision with correction. Case 19, a young woman, twenty-one years of age, was discharged with normal tension, but returned to the clinic ten days later with a painful, blind eye; tension was fifty-five. After linear extraction the pupil was black but there was no light perception.

The other patient with postoperative complications was a child three years of age. The child's mother, a nurse, reported that the general condition was normal and the Wassermann test was refused. Syphilis was considered the cause of convulsions and iridocyclitis which followed the second operation on the right eye. Hemiplegia and facial paralysis also developed at the time of operation.

It was necessary to incise the secondary membrane in three cases.

Traumatic Cataract

Complete discission of the lens was the method of treatment employed in four cases of traumatic cataract. Two of these

patients presented postoperative complications. Patient No. 2 was hypersensitive to lens protein and was desensitized before the operation. However, he developed an endophthalmitis phacogenetica and a low-grade iritis. A mild iritis with the formation of incomplete posterior synechiæ was the only unfavorable postoperative reaction in another case (Case 11).

It was necessary to incise the secondary membrane in one case.

High Myopia

The results of complete discission in one case of high myopia were good.

In a number of the patients operated upon it was impossible to obtain the final vision from the incomplete clinic records.

Postoperative Visual Acuity

The results in twenty-four cases were as follows: In ten cases vision was improved in varying degrees from 20/200 to 20/20; in three cases it could not be improved above 20/200; vision was unchanged in two cases; three operations were performed on children whose vision could not be determined; five cases were clinic patients who were not followed up; and one patient became blind.

Summary

1. The method of operation for congenital and juvenile cataract assumes greater importance after consideration of statistics which show that congenital cataract is the cause of blindness of 14.8 per cent of the pupils in schools for the blind in the United States. In one New York school for the blind ten per cent of the inmates have been unsuccessfully operated upon for congenital cataract in both eyes.

2. The danger of hemorrhage from an incision through the vascular limbus and the slightly increased technical difficulty are offset by greater safety from infection assured by the subconjunctival incision.

3. The single straight incision is a simpler technical procedure than the inverted V-shaped incision. It may be performed with less traumatism and the results are apparently as good as with the incision advocated by Ziegler.

TABLE III — *Resul*

Number of Patient	1	2	3	4	5	6	7	8	9	10	11
Age	8	17	17	3	13	13	14	17	17	26	2½
Sex	F	M	F	M	M	M	M	F	F	F	M
Diagnosis: 1. Congenital Cataract	O.U.		O.D.	O.U.	O.D.	O.S.	O.D.	O.D.	O.S.	O.S.	
2. Traumatic Cataract		O.D.									O.S
3. Nystagmus	O.U.		O.D.								
4. Aniridia											
5. Aphakia											
6. High Myopia											
Date and Type of Operation and Eye Operated Upon:	O.D.	O.D.	O.D.	O.D.	O.D.	O.S.	O.D.	O.D.	O.S.	O.S.	O.S
1. Iridectomy			8/15/27	6/18/27			3/18/25				
2. Needling Prior to Complete Discission			9/27/27		8/31/26						
3. Complete Lens Discission	3/27/28	2/5/27	2/28/28	6/30/25	12/17/28	6/18/27	6/22/25	2/4/28	9/1/28	2/4/28	11/5/
4. Paracentesis and Removal of Soft Lens Matter						7/ 7/27*					
Complications: 1. Hypertension						O.S.					
2. Hypotonia with Iritis		O.D.									O.
3. Hyphemia				O.D.							
4. Incarceration of Iris						O.S.					
5. Endophthalmitis Phacogenetica		O.D.									
6. Phaco-anaphylaxis					O.D.						
Condition of Lens: 1. Secondary Membrane Without Opening	O.D.			O.D.	O.D.						
2. Good Opening in Membrane		O.D.	O.D.						O.S.	O.S.	
3. Partial Absorption of Lens						O.S.	O.D.	O.D.			O.S
Vision Before Operation: O.D.	2/200	18/200	2/200	———	20/200	———	20/70	L.P.	———		———
O.S.	20/200	20/30	20/100	———	———	20/200	20/100	———	4/200		———
Vision with Correction After Operation: O.D.	15/200	20/25	4/200	———	20/70	———	20/50				———
O.S.	20/200	20/20	20/100	———	———	20/200	20/50				———

10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	TOTAL	REMARKS
26	2½	15	18	18	4	4	25	17	21	7	17	3	18	29		
F	M	F	M	M	M	M	M	M	F	M	F	F	F	M	M-14; F-10	
.S.			O.D.	O.S.	O.D.	O.S.		O.U.	O.U.	O.U.	O.D.	O.S.		O.U.	19	
	O.S.	O.S.					O.D.								4	
															2	
			O.D.	O.S.											2	
											O.S.				1	
													O.U.		1	
.S.	O.S.	O.S.	O.D.	O.S.	O.D.	O.S.	O.D.	O.D.	O.S.	O.S.	O.D.	O.S.	O.U.	O.D.		
		11/9/25													4	Case 3—Recession O.U. 3½ mm. because of esotropia.
														1/19/29 2/ 2/29 2/13/29	5	
4/28	11/5/27	11/16/25	1/2/21	1/22/21	5/29/31	1/2/31	7/9/29	1/29/32	1/29/32	12/11/31	2/19/32	4/15/32	1922	8/23/29	24	
								3/11/32*	2/17/32*						3	*Washing out of anterior chamber because of high tension in 3 cases.
								O.D.	O.S.		O.D.				4	Case 4—Following convulsions and iridocyclitis left hemiplegia and facial paralysis developed after the second operation, probably due to luetic disease of the brain.
	O.S.														2	
															1	
															1	
															1	
															1	Case 5—Was desensitized before operation.
		O.S.													4	
.S.			O.D.		O.D.	O.S.	O.D.	O.D.		O.S.	O.D.	O.S.		O.D.	13	
	O.S.			O.S.					O.S.						6	
	—— ——	20/30 L.P.	L.P. ——	—— 5/200	20/200 ——	—— 20/200	5/200 20/20	20/70 20/70	20/70 4/200	20/70 H.M.	4/200 20/30cc.	—— ——		16/200 20/50		Case 3—Choroidal changes in right fundus. Case 19—Returned in 3 weeks with a painful, hard and blind eye.
	—— ——	20/30 L.P.	20/200 ——		20/50	20/50	20/100 20/20	20/30	No. L.P.	20/70 20/30	20/30 20/30	—— ——	Vision greatly improved	20/40 20/50		Case 24—Deposits on Descemet's membrane before operation which remained unchanged after operation.

4. If the iris is widely dilated with atropine and epinephrine, the subconjunctival incision may be made as easily as when an iridectomy has been performed. Preliminary iridectomy seems unnecessary in patients under ten years of age, provided a thorough preoperative examination has been made.

5. Complete discission of the lens by a single straight incision in rabbits and guinea pigs resulted either in complete or partial absorption of the soft lens matter, while anterior capsulotomy by a single straight incision invariably led to scar formation in the anterior capsule, and a secondary operation was always necessary.

6. Increase of tension and inflammation of the eye with hypotonia was observed in approximately the same number of the experimental animal eyes, irrespective of the type of operation.

7. The lens of the rabbit subluxates easily and is so thick that complete discission is more difficult to perform than on human eyes. Guinea-pigs' eyes, because of the size and dark color, are not suitable for experimental complete discission or partial discission of the crystalline lens.

8. In twenty-four operations on human eyes there were two serious complications. Severe iridocyclitis and left hemiplegia developed in a child with unrecognized syphilis. This complication could possibly have been avoided if the Wassermann test had been made. In one case hypertension was the cause of complete blindness because the patient did not return to the clinic and the lens matter was not expressed early enough. Increased tension in two other cases necessitated linear extraction. It was necessary to incise the secondary membrane in four cases.

9. The advantages of complete discission over anterior capsulotomy appear to be: Fewer operations are usually required, thus reducing the danger of infection; reaction is less and hypertension is less likely to occur.

Conclusion

Complete discission of the crystalline lens has afforded good results in the treatment of congenital, juvenile and traumatic cataracts and in high myopia. It would be unfortunate if, because of adverse criticism, this procedure were to be discarded without further trial.

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DISCUSSION

Dr. E. C. Ellett, Memphis, Tenn.: Since the publication in 1921 of Ziegler's paper on through-and-through discission of the lens for soft cataract, I have made use of the method as the opportunity presented with results that have seemed to me more satisfactory than those obtained by the method I had previously employed. The final visual result is probably not affected, but the solution of the lens occurs with less disturbance and less subsequent interference than formerly. My experience is too small to enable me to say that it occurs any more rapidly.

The advantages of this method seem to me to be that the lens has more room to swell and that secondary glaucoma is less apt to occur and simple linear extraction less often required. The table which I show gives the main points in twenty-six eyes.

My experience agrees very well with that of the author of this paper. In spite of the advantages that pertain to a puncture made through the conjunctiva and sclera, the blade of the knife-needle is then more nearly parallel to the surface of the lens than when the puncture lies in the cornea. The lens, therefore, is not so easily entered and the danger of dislocating it is measurably greater. The same objection applies to multiple and V-shaped incisions, and for this reason I have adopted the single free deep cut as described by Dr. Berens, and I prefer to enter the knife through clear cornea. In brief, the method as described by Dr. Ziegler seems to me distinctly superior to the method of puncture, followed by freer needlings which often need to be repeated and often necessitate linear extraction.

Name	Age	Number of Discissions	Linear Extraction	Capsulotomy	Visual Result: Metric
D. P.	(2)	1	0	0	P. L.
G. J.	(3)	1	0	0	?
R. W.	(19)	1	0	0	2/60
R. W.	(19)	1	1	0	6/7.5
M. B.	(2)	1	0	1	5/30
M. B.	(2)	1	0	1	5/30
L. N.	(1)	1	0	1	Sees very well
L. N.	(1)	1	0	1	Sees very well
V. M.	(11)	1	1	0	6/30 (incomplete)
E. G.	(15)	1	0	0	Incomplete
P. R.	(1)	1	0	1	Sees very well
P. R.	(1)	1	0	1	Sees very well
H. S.	(6 mo.)	3	0	2	?
H. S.	(6 mo.)	1	0	2	?
L. T.	(29)	1	1	0	5/7.5
L. T.	(29)	1	1	1	5/5
J. T.	(3)	1	0	0	6/15
J. T.	(3)	1	0	1	6/6
B. B.	(23)	1	1	0	0
R. C.	(14)	1	0	0	6/6
B. L. D.	(5)	1	0	1	5/6
B. L. D.	(5)	1	0	2	5/7.5
E. B.	(28)	1	1	0	6/6
E. B.	(28)	1	1	1	6/6
L. B.	(5)	1	0	2	6/7.5
L. B.	(5)	1	0	1	6/12
26		28	7	19	

Dr. Meyer Wiener, St. Louis: I want to congratulate the essayists on the excellence of their paper, for in my experience the reaction is certainly very much less with Ziegler's operation than with the puncture of the anterior capsule which we formerly did. I have been doing the Ziegler operation since long before it was published, because Ziegler performed this operation at least eight or ten years before he first published it, and I saw some of the first operations he did.

One point I have found of value is this: Sometimes after the discission, though rarely after the Ziegler through-and-through discission, we have found the tension increased; this we have been able to relieve with glaucosan or adrenalin packs. My experience with glaucosan has not been favorable in glaucoma, but this type of case is an exception.

One type of case which Dr. Berens did not mention is where there is complete posterior synechia in congenital cataract. In that case I think it is absolutely essential that the iris be nicked and that there be a V-shaped opening including the lower border of the iris in both punctures as well as going through the lens. I have never seen any objection to this technique.

Dr. V. M. Hicks, Raleigh, N. C.: My interest in this procedure dates back to 1920, at which time it was my pleasure to work with Dr. Berens. In 1924 I reported a series of cases before the Southern Medical Association in New Orleans. I think the essayist has very well covered the ground, but I have three suggestions I would like to add.

First, I think the importance of complete mydriasis cannot be overemphasized, and in this case we have favored scopolamine over atropine because we have found that in a number of cases the continued use of atropine has given an allergic reaction.

Second, the lens is cut very easily with a Ziegler knife in a sawing fashion, but I believe it is important that the cutting occur only on the downward stroke of the knife. In a few cases I have completed a crucial incision, but the horizontal cuts were made only through the anterior capsule. This seemed to hasten absorption and there was no evidence of unusual reaction.

Third, I do not believe that the success of the operation should rest on the basis of vision. In considering the success of the procedure only the amount of reaction produced and the facility with which the lens is absorbed, together with injury to the other intraocular structures, should be considered.

We all know that in a goodly number of these children, because of previous degeneration in the posterior segment of the globe, good visual result is not secured. The type of operation selected cannot possibly alter this pathological complication. Our experience with this operation has come from eleven years of private practice and in doing the operating in the North Carolina schools for the blind during this period.

Dr. George Slocum, Ann Arbor: Aware as I am of the fact that Dr. Ziegler's discission operation has been frequently criticized, it is very gratifying to me to learn of Dr. Berens' favorable experience.

For a number of years, indeed since Dr. Ziegler's operation first came to my knowledge, I have been practicing through-and-through discission for soft cataracts, and the results have been so uniformly satisfactory that I have never returned to the older technic. In the first cataract I operated by this method there was no reaction; the pupil was more easily kept dilated, absorption was more rapid, there was no secondary glaucoma, and a second needling was unnecessary. In subsequent experience a second needling has been done frequently, but the other advantages first observed have been manifested in practically every soft cataract so operated. Perhaps I have been fortunate, but in my cases there have been no complications. I have not done a preliminary iridectomy.

From the first I have used the single vertical incision advocated by Dr. Berens, because the second incision seemed unnecessary as well as more difficult. Occasionally, in thin or deformed lenses showing unusual anomalies of development, I have considered the V incision advisable. This, however, has not been necessary in lenses not showing these peculiar anomalies. I wish also to commend the safety of Dr. Berens' subconjunctival puncture. It is my practice to puncture the cornea through the conjunctival limbus, and I have had no infections.

On the whole, my experience with the Ziegler operation has been so happy that I would be very unwilling to return to graduated needlings, sometimes complicated by iridocyclitis or secondary glaucoma, with the necessity for occasional linear extractions, undertaken either for the relief of the glaucoma or because of long, slow and tedious absorption.

Dr. Conrad Berens, closing: Dr. Ellett raised the question of dislocating the lens by the incision at the limbus. We have caused subluxation of the lens in one case, and the operation should not have been attempted. The patient was eighteen years of age, and another type of operation should have been done. As a matter of fact, it is possible that complete discission should be done only on young children and I shall probably limit it to patients twelve years of age or under, depending on the type of cataract found.

Dr. Wiener uses glaucosan and epinephrine packs to control the tension. I have used epinephrine pledgets combined with instillation of a three per cent solution of ephedrine several times a day, with excellent results in certain cases. He spoke of the advisability of using a V-shaped incision when posterior synechiæ are present. This seems to be an excellent plan in these special cases, but the necessity for it has not occurred in the cases I have seen.

Dr. Hicks also spoke of the importance of an age limit and mentioned ten years. He pointed out the necessity for obtaining complete mydriasis. If epinephrine is not used in combination with atropine the iris contracts when the anterior chamber is opened and it is difficult to perform the operation properly without nicking the upper border of the iris. He also stressed the importance of cutting through the lens only with the downward stroke. Do not try to cut as the knife is withdrawn, but as Dr. Hicks directs, and I prefer to use a gentle sawing stroke.

The incision Dr. Slocum spoke of is useful in certain cases and I think this is also an important addition to the technic of the operation. Certainly in my experience the advantages of complete discission outweigh the disadvantages in selected cases, and I should not like to be forced to return to the older methods of operation.

VISUAL FIELD STUDIES

Pseudo Upper Form Field Contractions

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IN ANY BRANCH of medicine during the routine examination of a large series of cases it frequently happens that some relatively unimportant or pathologically insignificant condition recurs with sufficient frequency to attract attention. The analysis of cases presenting such a condition, using as a background all of the available clinical and laboratory data, will occasionally bring to light correlations which may assume an etiological significance. With this fact in mind the writer desires to report a series of cases all of which presented a cutting of the upper form (or white) field of vision with recovery to normal limits on raising the upper eyelid.

The cases constituting this series, 230 in number, were seen in the routine eye examination of 1,903 patients in a group of 3,100 studied at the Evans Memorial of the Massachusetts Memorial Hospitals in connection with the endocrine service over a period of five years ending December 31, 1928. No selection of cases was made as far as the ophthalmologist was concerned, the eye examination being one of many routine procedures carried out on all house cases regardless of any complaint and on a large number of out-patients when it seemed helpful to obtain the eye findings as an aid in the diagnosis of a wide variety of morbid states.

All of these patients were admitted for more or less complete diagnostic study, the general method of which has been described elsewhere.[1] The ophthalmic data observed and recorded included the determination of visual acuity, pupillary measurements and reactions, extraocular motions, the condition of the ocular media and fundi, and the determination of the form and color fields.

TABLE 1 — *General Diagnosis*

Endocrine Pituitary	
Hyperfunction	2
Dysfunction	47
Hypofunction	20
Thyroid	
Hyperfunction	1
Dysfunction	4
Hypofunction	30
Gonad, hypofunction	13
Pancreas, hypofunction	1
Pluriglandular, dysfunction	8
Total Endocrine Cases	126
Non-Endocrine Infections	
Sinusitis	3
Tuberculosis	5
Arthritis	4
Focal (miscellaneous)	6
Psycho-Neuroses	
Neuroses	4
Psycho-Neuroses	5
Psychoses	1
Cardio-Vascular	
Cardiac	4
Renal	5
Cardiac-Renal	8
Hypertension	1
Lesions of Central Nervous System	18
Hepatic	19
Syphilis	8
Goiter, Non-Toxic	5
Blood	3
Miscellaneous	
Skin	2
Pelvic	1
Cancer	1
Gastro-Intestinal	1
Total Non-Endocrine	104
GRAND TOTAL	230

Table 1 gives the general diagnosis covering quite a wide range of conditions. One point stands out as significant in this group, namely, the frequent evidence of general, metabolic

disturbance. That this should be anticipated in the endocrine group follows directly on the fact that the endocrine glands are primarily regulators of metabolism. In the non-endocrine cases there are likewise many unmistakable evidences of profound metabolic disturbance. This state and the peculiar eye findings are the only two common points presented uniformly throughout the series.

In this same group of cases were discovered an unusual number of markedly contracted fields which were reported in 1929.[2] Since the findings reported in this communication have not been dealt with particularly by authors of text books or investigators and since their occurrence is quite frequent, especially with certain types of field-taking devices, it seemed warrantable to the writer to offer his findings with an attempt to explain their significance.

The Peter hand campimeter was used in recording the fields. This apparatus was selected for its flexibility and adaptability to the varying conditions under which many of these patients required examination. Many were bed patients and many had physical or mental faults or were of an age which made them unsuitable for the more extensive or laboratory procedures which might have counted in suitable cases for greater accuracy. The number of patients tested at a given session also entered into the selection of this device. Often ten or more patients were taken consecutively. The frequent fatigue of patients studied in many instances was an argument in favor of a quickly-outlined visual field.

The technique consisted in having the patient (without glasses) comfortably seated in an armchair resting his elbows thereon and holding the slate with each hand in such a position that he obtained parallelism between the campimeter and the general facial plane, and at right angles to the line of fixation. A small white cross was made in the center of the slate in which center was made a small red dot, it being much easier thereby for both patient and examiner to secure and maintain reliable fixation in the average case. The blind spot was first delineated, using one-degree white object, and passing from blind to seeing. The form field was then obtained by a two-degree white object, in slight motion, passed in over several meridians. The field for form rather than for white was considered more reasonable in

the absence of a standard illumination and with lowered visual acuity and other factors present in many patients which will later be discussed. Color fields were outlined by the same two-degree objects. Good daylight or approximately equal diffuse artificial illumination was used. For a first study or general field delineation these larger test objects were used. When pathologic deviations were encountered re-studies were made with smaller objects and frequently on the more exact Ferree-Rand apparatus.

These upper form field cuttings are named by the writer *pseudo-form field cuts* or *contractions* when the field was found normal on raising the upper eyelid by the examiner's finger. The cutting varied from a broad band of twenty degrees extending from the temporal to the nasal limits of a more or less quadrangular form to a nearly triangular loss when a lid drooped over the outer palpebral fissure. No cases were included which recorded less than a five-degree cut and a full temporal to nasal loss.

TABLE 2 — *Upper Form Contractions Recovered by Raising Lids*

230 Cases in 1903 Patients (12+%)

Form contractions only	224 cases, 97.4%
Form and Color contractions	6 cases, 2.6%
Binocular contractions	202 cases, 87.8%
Monocular contractions	28 cases, 12.2%
Also Blind Spot enlargement	60 cases, 26.0%
Pupil, Muscle, Media, Fundus changes	47 cases, 20.4%
Refractive errors (estimated)	105 cases, 45.6%

Table 2 shows that a large percentage, 87.8 per cent, were bilateral losses although not always symmetrical. A few, 12.2 per cent, of the patients exhibited monocular cuts, some of which were due to trauma. A few, 2.6 per cent, had cuts deep enough to invade the normal fields for blue and occasionally also for red. The forty-seven cases exhibiting deviations from normal in pupils, extraocular muscles, media, and fundi, should not be considered as affecting the upper field limits by these faults since recovery was obtained by raising the lid. However, refractive errors seemed to play a considerable part in producing this phenomenon. The effort at fixation necessary in presbyopia, perhaps less so in high hyperopia and marked astigmatism, especially at the short distance of the Peter campimeter, certainly lead the writer to this conclusion.

TABLE 3 — *Classified Lid and Orbital Conditions*
136 Cases

Orbital encroachment	2
Deep-set eyes	8
Overhanging brows	3
Long lashes	2
Heavy or redundant lids	4
Dermatitis	1
Blepharochalasis	3
Enophthalmos	1
Ptosis (trauma homolateral 1, contralateral 1)	6
*Lid droop	106

* Scarcely noticeable unless sought for.
Many refractive errors.

Table 3 analyzes the attempt to find some organic or functional factor having etiologic significance. Previous perimetric and campimetric experience had not brought this condition so noticeably to the writer's attention until the work at the Evans produced a large volume of consecutive material under good control from the point of view both of the data obtained and the records of the same.

The first interpretation of this phenomenon was that it might represent a functional fatigue syndrome similar to the quadriceps test suggested by Lahey[3] in cases presenting thyroid dysfunction and deriving from levator lag or fatigue. Analysis of the first series of cases, however, showed so large an incidence in patients presenting disorders unassociated with thyroid disease that no specific diagnostic significance could be attributed to it. At this juncture greater notice was given the conditions listed in this tabulation which accounts for the fact that only 136 cases were thus analyzed.

Holloway and associates[4] found wider form limits in exophthalmos of hyperthyroidism which might be expected in contradistinction to the one case of enophthalmos in this series. Ptosis would logically be expected to register upper field limits. Figures 1A and 1B show such a condition and it is suggested that the visual field might be a satisfactory method of measuring and recording the amount of ptotic fault. Anatomical interference to a well-opened palpebral fissure is easily accounted for by changes in the bony orbit, tissues of the brow and upper lid and even by long lashes. Pathologic changes in the lid tissues due to dermatitis, blepharochalasis, or edema act similarly. But in this series

the largest number of faults were found in cases where lids were apparently normal both organically and functionally. Nevertheless, on careful observation, it was noted that the lower border of the upper lids covered the upper pupillary area, thus interfering with full upward vision. For want of a better term these were classified as *lid droop*.

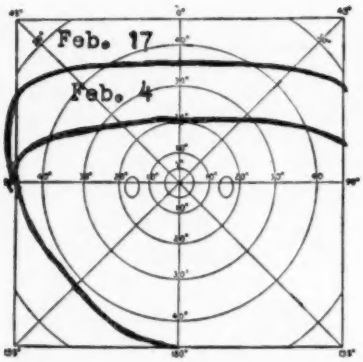


FIG. 1-A—Functional Ptosis measured and recorded at intervals.

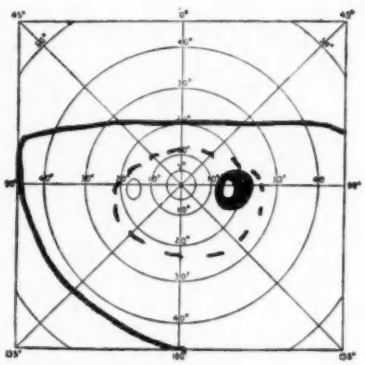


FIG. 1-B—Organic Ptosis. (Contralateral eye recovered a normal field from and equal cut by fascia lata hammock operation by Dr. George Derby).

In a previous paragraph refractive errors were referred to in relation to their effect upon these upper field contractions. This is easily demonstrated in many cases by observing first the level of the upper lids when the patient fixes at a distance and then fixes at the near point upon an object about the same size as that on the campimeter or perimeter and placed at a similar distance and level. It will then be noted that a decided lowering of the lid level transpires. This is doubtless due to an associated orbicular tone synchronous with fixation in accommodation. The physiologic explanation may not be apparent but the clinical observation is a fact. Refractive errors, undoubtedly, are responsible for many of our findings when the field is taken without glasses. Figures 2A and 2B demonstrate the larger field with glasses. Figure 3A shows a campimeter pseudo-contraction verified on the Ferree-Rand instrument.

One of the outstanding findings elicited in a careful study of these patients was the metabolic faults determined and variously exhibited as previously referred to in Table 1. The low-

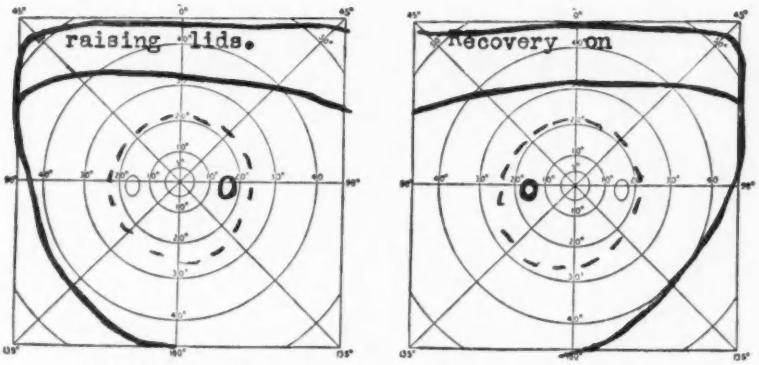


FIG. 2-A—Fields taken without glasses. V.R.—20/100 : V.L.—20/100.

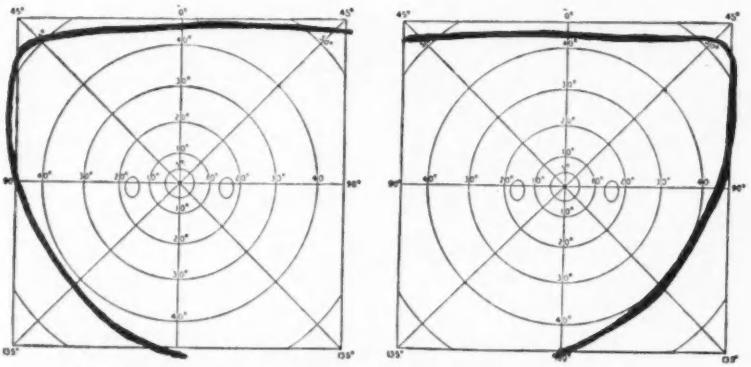


FIG. 2-B—Same fields taken with reading glasses:
O.D. + 2.25 D.S. = + 2.00 Cax 105

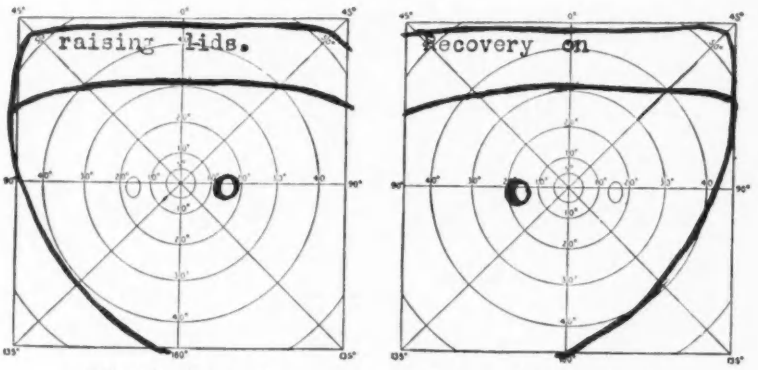


FIG. 3-A—Recovered upper form contractions. Peter Campimeter.

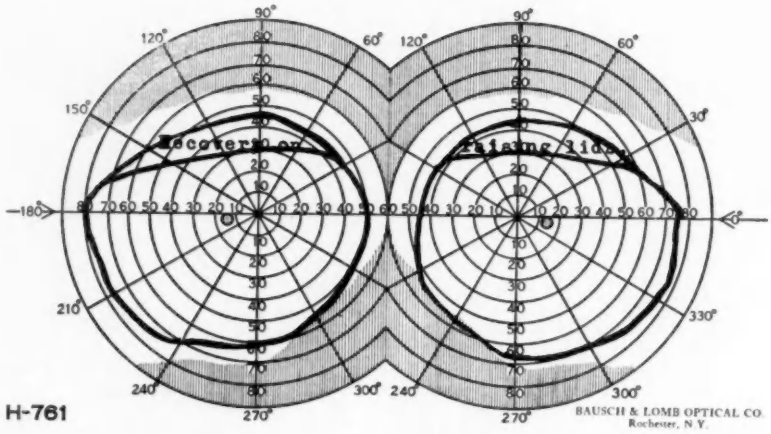


FIG. 3-B—Pseudo upper form contractions of 3-A verified on Ferree-Rand perimeter.

ered physical and also, to a lesser degree in many cases, the lowered mental tone or alertness resulting therefrom reflects itself in no small degree in body attitudes and facial expression of which latter the eye-lid muscles have much to do with the picture. In a normally prominent eye or in exophthalmos, the field is unusually wide in its limits, and per contra anything which narrows the palpebral fissure in any way may contract the field proportionately in its altitudinal limits.

In considering the field defects produced by observed conditions in this series of cases, it comes to mind that many other conditions logically could produce like phenomena. Edema of the upper lid from any cause such as insect stings, lid abscesses, empyema from frontal or anterior ethmoid sinusitis, orbital cellulitis, myxedema of hypothyroidism; restriction of motion by nodules or induration as in chalazia, ankyloblepharon, symblepharon, scars from trauma, burns or suppurating sinuses; levator palsies and fractures of the upper anterior orbital margin with depression — all these are to be thought of. Morton[5] in the Encyclopedia of Ophthalmology discusses many factors influencing the field of vision. A larger pupil gives a wider field by light reaching the anterior areas of the retina. In accommodation, as in hyperopia, the iris plane is further forward and since the nodal point follows this movement the field is widened thereby. Conversely, smaller pupils or myopia will register proportionately smaller visual fields. Cranial configuration and

bony orbital margins may interfere as well as deep set eyes in normal orbits. Some of these are racial characteristics. Redundancy of fat or skin in the upper lids restrict upper limits. He refers to several observers who analyzed the effect of correcting glasses on the peripheral fields. If eye-glasses are small, they either may not cover the extreme perimetric limits or affect the field limits by prismatic displacement, sometimes enlarging, sometimes contracting, according to the exact physical problem involved for the given case. Nothing is noted in these reports concerning a wider palpebral fissure obtained by more easy fixation when the refractive error is great enough to produce "squinting" effort and which the writer of this communication considers of some importance, especially for short range apparatus.

Ferree and Rand[6] in analyzing a large group of patients to determine the effect of errors of refraction, age, and sex in relation to the size of the form field conclude: Refractive errors are influencing factors varying with individuals, more important after the age of forty, and sex plays an insignificant part. Their chart shows a considerable narrowing in the presbyopic age, even with their ideal apparatus.

Peter[7] calls attention to the width of the palpebral fissure in relation to possible altitudinal contractions; to the effect of pupillary diameter and plane of the iris; and to the activity of the extreme nasal retina as well as its forward attachment resulting in wider temporal limits. He states that the field is narrowed in myopia and enlarged in hyperopia. In presbyopia the field is generally narrowed because of reduced retinal sensibility resulting from subnormal accommodation.

Traquair[8] discussing the same factors feels that uncorrected refractive errors may cause apparent contraction of fields.

Roemer[9] in discussing the interference of lids in the visual field states that if the upper lid is strongly raised or drawn back, it will result in wider limits.

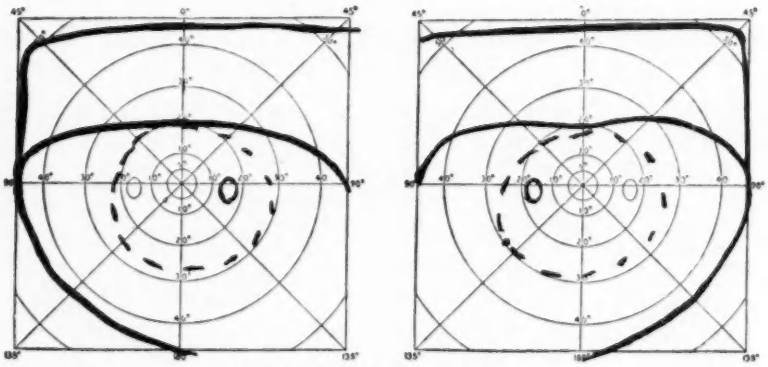
Since this study concerns itself only with upper contractions and normal limits obtained by raising the patient's upper lid, at least some of the factors referred to as limiting visual fields by the writers before mentioned do not enter into this particular problem. These various orbital or lid conditions are infrequently encountered in cases in which field studies are car-

ried out for specific diagnostic purposes but when a large series of patients are routinely studied, regardless of an eye complaint or where ophthalmologic faults are not expected, the relation between the phenomena and the apparent etiology becomes more apparent as is seen in this study.

Two brief protocols exemplifying one endocrine and one non-endocrine condition may be expository to the text.

CASE B-803 — *Thyroid Failure*

The patient was a white boy sixteen years of age complaining of pain and discharge in the right ear. There was a history of ear trouble since early childhood with bilateral paracentesis at the age of two during an attack of pneumonia. There had been frequent recurrent attacks of pain and tenderness. No marked loss of hearing. The patient had had several attacks of pneumonia and several tonsillectomies. He had had hives in the previous spring. The boy was fairly nourished, there was a thick grayish discharge from the right ear with the drum partly occluded, there was much wax in the left ear, the nose showed inflamed membrane and crusting. There was some rigidity and tenderness in the upper right abdominal quadrant. The urine was substantially normal except for a trace of albumin. There was a marked lymphocytosis, and depressed sugar tolerance. The boy was 10 per cent underweight, the basal metabolic rate — 19 per cent. The pulse was slow; roentgenograms of the sinuses and mastoids were negative. The Barany reaction was normal. A subsequent abdominal examination did not confirm the earlier report. An audiogram shows slight loss of



July 20, 1927—Form, 2°; Red, 2°.

hearing in the right ear. A duodenal function test showed marked hepatic disturbance.

The general picture in this case was consistent with thyroid failure with the sole exception of the lowered sugar tolerance and this was directly attributable to the demonstrated hepatic dysfunction.

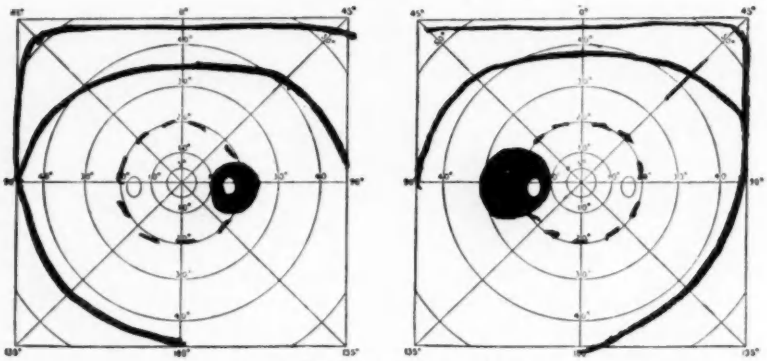
Eye Findings: Vision 20/20 and Jaeger 1, each eye. Pupils and extraocular muscles normal. Media and fundi normal. Fields exhibit marked upper form cutting recovered by raising the lids which cover the upper third of pupils.

CASE S-1509 — *Pulmonary Tuberculosis*

The patient was a white woman twenty-five years of age complaining of weakness, loss or weight, easy fatigability, and a marked susceptibility to colds. She dated the condition from an attack of influenza two years previously and the whole condition became progressively worse. There was also a history of pain along the lower spine which was intermittent in character. She had had pneumonia some years previously and there was a history of aural abscess following a tonsil and adenoid operation. She complained of a chronic cough, late onset of the catamenia with earlier irregularity but present normal function. She had lost five pounds in the past two weeks. Examination showed a flushed face, a somewhat flat chest with a generalized distribution of coarse and moist rales. The urine picture showed scanty elimination and high residual nitrogen. The blood was lymphoid in type. She was 20 per cent below her predicted weight and exhibited some loss of lung volume. The basal rate was the normal value of —1 per cent with a somewhat rapid pulse. Roentgenogram of the chest showed slight mottling at both apices above the clavicles, diffuse increased density throughout both lungs except at the bases.

The patient gave no evidence of any endocrine condition, while both the physical findings and roentgenograms indicated active pulmonary tuberculosis.

Eye Findings: Vision was 20/20 and Jaeger 1, each eye. Pupils and ocular movement were normal. Media clear. Fundi were normal except for yellowish discs. Visual fields show slight upper form cuts recovered by raising the lids; slightly contracted color fields; quite large blind spots.



June 1, 1927—Form, 2°; Red, 2°.

Conclusions:

1. Upper field contractions may be due to a wide variety of conditions affecting the lids, orbits, intraocular structures, optic nerves or the visual pathways.
2. If normal field limits are obtained by a wider palpebral fissure, the *pseudo - contraction* differentiates the phenomenon from altitudinal hemianopsia.
3. A careful study of the patient may also discover metabolic or other faults having a contributing bearing upon the field change.
4. Perimetry is suggested as a means of measuring and recording ptosis.

Grateful acknowledgment is made to Allan Winter Rowe, Ph.D., director of the Evans Clinic for an analysis of the physical and laboratory data, and to the writer's associates for aid in the ophthalmologic observations and records.

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DISCUSSION

Dr. John N. Evans, Brooklyn: The paper which Dr. Rowland has presented is of distinct value in calling our attention to the upper field periphery. The greater interest in the lateral hemianopsias and in the classical central field changes of glaucoma, etc., has directed our attention from a region which is more productive of valuable data than one would at first be inclined to suspect.

Dr. Rowland has pointed out the influence of surrounding parts which must be considered in the interpretation of results. The early writers on perimetry showed great ingenuity (Am. Encyclo. Oph., Vol. XII, p. 9433) in avoiding false conclusions through encroachment of adjacent structures.

If an unselected group of fields be taken from our files, a certain number will show superior defects which are not always easily explained. I reviewed some of my own records for material for this discussion, but was forced to discard all of them because the angle of head fixation had not been accurately noted. Conditions were therefore not reproducible for comparison. In normal subjects lid position bears a relation to the visual line, which relation changes not only with eye position, but also with head position. This whole question is very much more complex than one might expect, particularly when we recall the elaborate studies on postural reflexes by Magnus, de Kleyn, and a host of others (Duke-Elder, pp. 615 and 619), (Parsons' Perceptions). More complicated yet must be the factors which influence these relations in patients who are suffering from various diseases and in whom the reclining, sitting or standing postures can conceivably produce wide variations in the lid position and head angle. One might think that the headrest of the Ferree-Rand perimeter would be sufficient guarantee of an ideal position, but the facial angle for a small series of cases examined in this headrest was such that the head was tipped forward fifteen degrees. Thus in a certain number of normal subjects there would be a 1 mm. lid droop, which might not be present if head fixation were corrected.

As regards the effect of elevation of the lid. Dr. Edward Jackson measured the effect of lid elevation on two hundred patients (Trans. Sec. Ophth. A.M.A. 1921, p. 73), and presented very definite figures to show that in all but seven of the cases the eye moved forward when the lid was raised. This protrusion of the globe was .75 mm. in most cases, but varied from .25 mm. to 1 mm. It was practically absent in people after sixty years of age. Dr. Jackson apparently referred to elevation of the lid by voluntary action of the lid muscles, but it must be obvious that digital or instrumental elevation of lids would have to be most carefully performed so as not to disturb the position of the globe by direct or reflex action. Apparently infinitesimal changes in the prominence of the globe would make relatively great variations in the peripheral field boundaries.

From these few points it becomes at once apparent that the interpretation of the upper peripheral field can only be made after careful consideration of a great variety of factors.

OTOLARYNGOLOGIC SESSIONS

THE PRACTICAL APPLICATION OF WITTMAACK'S THEORY OF PNEUMATIZATION

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SINCE Wittmaack first expounded his theory on the mechanism responsible for the pneumatization[1] of the temporal bone a great deal has been written on this subject. More and more proof has been brought out until now one can accept as fact the principles of the normal process of pneumatization which Wittmaack has set forth. In brief, these are as follows.

The mastoid process of the newborn child, when seen histologically, appears as a spongy bone showing fatty bone marrow filling the spaces between the bony trabeculæ. The middle ear at birth is completely filled by a high embryonal type of myxomatous connective tissue which lies immediately under an epithelial covering. In the conversion of the embryonal tissue into its adult form there occurs a gradual shrinking and contraction of this tissue which eventually becomes the adult subepithelial tissue found everywhere underneath the epithelial lining of the ear and the mastoid cells. As soon as the child takes its first breath of air this myxomatous subepithelial tissue begins to grow from the antrum and epitympanic space into the adjoining marrow spaces located in the mastoid cells. In the course of time this embryonal connective tissue completely replaces all of the fatty bone marrow. This is the first step in the process of pneumatizing the temporal bone. As this embryonal tissue then undergoes its normal contraction to eventually become adult connective tissue it causes its epithelial lining to be drawn inward into the intertrabecular spaces. This constitutes the second step in the process of pneumatization. Finally, when the contraction of the embryonal tissue has been completed, the epithelium will be found to line the bony trabeculæ within the mastoid process. Thus there results a series of spaces bound by the bony trabeculæ and lined by epithelium under which is the adult form of con-

nective tissue. These spaces all communicate with the antrum and are filled with air.

This is the normal process of pneumatization. It occurs in every one in whom no interference with the normal process is presented. This explanation of Wittmaack's meets with practically no opposition. On the other hand, his explanation of what occurs when the normal process of pneumatization is interfered with has been subjected to a certain amount of criticism by some observers.[2 3 4] However, Wittmaack has been able to show by histological study, and has been substantiated by many observers along anatomical, clinical and roentgenological lines of investigation, that the result of an interference with the normal processes of pneumatization causes a failure of the mastoid process to become pneumatic.[5 6 7 8 9] There are two main types of mastoid processes which will result from an interference with pneumatization: the infantile or diploic and the sclerotic mastoid process. However, depending upon the stage at which the normal pneumatization is interfered with, a mixture of types within the mastoid process may result.

If an uninfected foreign body, such as meconium, vernix caseosa or vomitus, enters the middle ear through the eustachian tube before the process of pneumatization has been completed, there will occur a disturbance of this process. The irritation caused by the foreign body on the embryonal type of connective tissue will produce a hyperplasia of the subepithelial connective tissue and prevent the normal contraction of this tissue. Consequently, the ingrowth of this subepithelial myxomatous tissue into the marrow spaces of the mastoid process will not be followed by any attempt at contraction due to the hyperplasia which has been set up by the foreign body irritation. The first step in the process of pneumatization therefore occurs but it will not be followed by the second step; namely, the normal contraction and drawing in of the epithelial lining. This fibrous tissue which has replaced the fatty bone marrow will persist within the intertrabecular spaces and in the course of time will be subjected to the osteoplastic activity of the growing osseous trabeculæ. There thus occurs a metaplastic ossification and there then results a sclerotic mastoid process.

Should this type of interference with the normal process of pneumatization occur after a certain amount of contraction of

the subepithelial connective tissue has set in and a few pneumatic spaces have been developed, any further pneumatization will be stopped at that point and there will then result a mixed type of mastoid process showing partial sclerosis and partial pneumatization.

On the other hand, where a pathogenic micro-organism enters the middle ear and produces a suppurative infantile otitis media, the inflammatory lesion, after it has run its course, will be healed by an evidence of increased fibrosis in the inflamed tissue. This tendency to heal the suppuration by a production of fibrotic changes causes a very rapid contraction of the subepithelial myxomatous tissue. This then results in an enhancement of the contraction of the embryonal tissue. Consequently, if this type of infantile otitis should occur before the subepithelial tissue has grown into the mastoid process, such an ingrowth will never occur because of the rapid fibrotic contraction. There will then be, throughout life, a retention of the infantile type of mastoid process; namely, the spongy or diploic mastoid, because the pneumatization was interfered with before the first step in its development could take place.

If an infantile otitis should occur during the first stage of pneumatization, there would result a mastoid process which would show a combination of all three types; namely, one wherein there would be areas of cellular development, sclerosis and diploë.

I have accepted Wittmaack's explanation of the pathological pneumatization as the only one which logically explains the presence of sclerosis and diploë in adult temporal bones. Only on this basis can one account for the hitherto apparent discrepancies in our explanations of chronic purulent otitis media. Since the sclerotic mastoid process is the result of a failure of the bone to become pneumatized rather than, as formerly believed, the result of a disease which converts a pneumatic into a sclerotic bone, this sclerosis should not be viewed as pathological. That a sclerosis rarely, if ever, results from a chronic inflammatory lesion in a pneumatic bone has been demonstrated histologically by Wittmaack[10] and Beck.[11] I also wish to add histological proof of this. In one of the cases in my series, a child of seven, who had had a chronic otorrhea since the age of two, following scarlet fever, and on whom a radical mastoid operation was per-

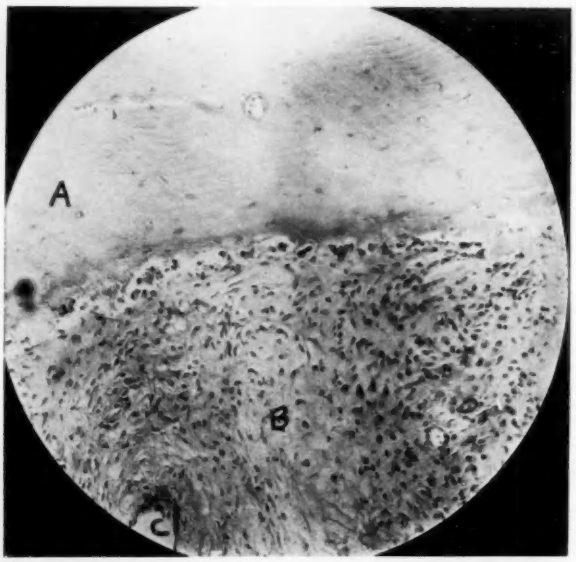

FIG. 1—Section of mastoid cortex and underlying cell space. A—Cortex. B—Connective tissue completely filling area between bony trabeculæ. C—Blood vessel.

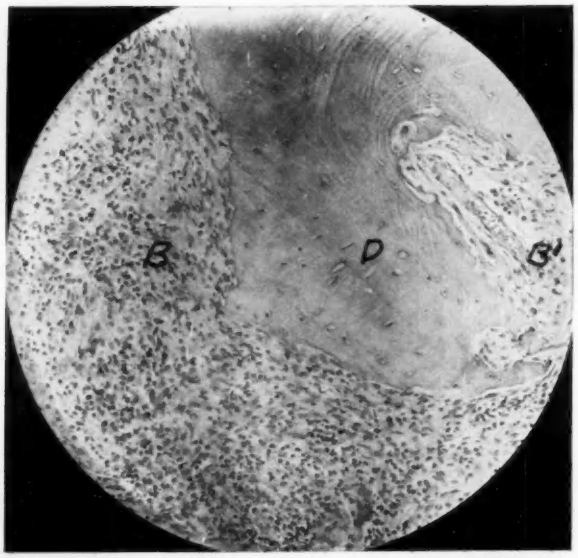

FIG. 2—Bony trabecula in depth of mastoid process. B and B'—Two interbecular spaces filled with connective tissue.

formed by me, the microscopic examination of the mastoid process revealed a normal cortex beneath which were bony trabeculæ containing dense connective tissue (Figs. 1 and 2). At no point in the section was there any sclerosis noted. In this we have evidence that where an acute necrotic otitis occurs during the course of scarlet fever in a temporal bone wherein normal pneumatization has taken place and wherein no surgical mastoiditis has developed, the cellular elements within the mastoid process which remain intact are not obliterated by any eburnation or sclerosis. Rather, the healing takes place just as anywhere else in the body; namely, by resorption of the exudate and replacement by connective tissue.

If we then take as our premise Wittmaack's teaching that eburnation or sclerosis found within a mastoid process is not pathology or the result of the pathological condition present in the middle ear, epitympanic space and antrum, we must conclude that there is no necessity surgically to remove this eburnated or sclerotic bone. Rather we must view the disease for which we operate as being confined purely to the middle ear and consider it solely as a disease of the middle ear. In those cases wherein the chronic purulent otitis media is due to bone necrosis the gross pathology always shows the disease as limited to the middle ear, attic and antrum. At operation, after the dense sclerotic bone has finally been entered there is always found a small space represented by the antrum from which the necrotic area can be seen extending into the epitympanic space and tympanum. Consequently, with this in view, I have within the past two years modified the technique of the radical operation in cases of bone necrosis to suit the individual case. Where the entire mastoid process is found sclerotic as determined by X-ray the surgery is limited to the antrum and tympanic cavity and whatever area surrounding the antrum may be diseased. The sclerotic bone occupying the remainder of the mastoid process is left in situ. The technique of the operation is no different than that described in the textbooks with the exception of the use of a drill to go through the cortex into the antrum and to smooth down the bony walls and the limitation of the operative procedure to the middle ear, antrum and whatever area surrounding the antrum is found diseased. This leaves a very much smaller area to be epidermatized and consequently shortens the after-treatment.

The attempt to individualize the radical mastoid operation and to limit the extent of the surgery employed in cases of chronic purulent otitis of the dangerous type is meeting the favorable consideration of other otologists. Rassudov,[12] Alexander,[13] Joseph,[14] Babbitt,[15] Smith,[16] and others advocate various procedures designed to limit the scope of the radical mastoidectomy, and have reported excellent results. The reason for the success of these measures is found in Wittmaack's teaching that sclerosis in a mastoid process is not a pathological condition which requires removal. Of seventeen ears which have been operated upon in the manner I have described, seven showed complete epidermization and cessation of the discharge four weeks after operation, five had a similar result within five weeks after operation, two within six weeks, one within seven weeks and one within ten weeks. The remaining case, one wherein a labyrinthine fistula was found, still has a discharge after three months due to a sequestrum and will require further surgery.

Another factor in Wittmaack's work which must be considered is his teaching that an inadequate escape for epithelial débris will result in the production of a cholesteatoma in a case of chronic purulent otitis media of the transformative type. Where a cholesteatoma is encountered as the cause of a chronic purulent otitis media, the same operative procedure is employed as previously outlined. The greatest percentage of cholesteatomas which came under our observation are secondary cholesteatomas and are the result of an ingrowth of squamous epithelium into the middle ear and antrum in an effort to replace the diseased tympanic mucosa with healthy epidermis. In a previous paper presented before this society[17] I endeavored to show that the epidermis played an important role in the repair of chronic suppurative otitic lesions. Only in those instances where a free exit for the desquamated epithelium was lacking did a cholesteatoma develop. The secondary cholesteatomas are the result of inadequate avenues of escape for the desquamated epithelium in the attic and antrum. The retention of this so-called cholesteatomatous material causes pressure upon the surrounding bone, a rarefying osteitis and a resultant enlargement of the cholesteatoma tumor mass. Consequently, since the matrix of the cholesteatoma is in reality nothing more than epidermis and since in the healing of a radical cavity the epidermis lining the membranous canal is utilized to line the radical cavity or a

graft of epidermis is employed, I feel that it is not necessary, except where invasion into the endocranium has occurred, to remove this matrix when performing a radical operation for cholesteatoma. Rather, this matrix should be conserved and utilized to line the radical cavity. The healing of the cholesteatomatous tumor and the prevention of its further growth will occur as soon as adequate drainage is afforded and a large opening made so that no epithelial débris can accumulate within the middle ear, epitympanic space or antrum.

In operating upon cases of cholesteatoma I have limited the radical procedure by entering the cholesteatomatous cavity through the mastoid cortex, removing the accumulated epithelial débris within it, lowering the posterior meatal wall to an extent which will permit adequate drainage into the external canal, removing the diseased ossicles where present, removing the polypi and epithelial débris in the attic, but retaining the cholesteatomatous matrix everywhere. In five cases of cholesteatoma upon which this procedure was followed two had a complete cessation of discharge with complete epidermization in four weeks, two in five weeks, one in six weeks. All these five cases have since been under observation, the shortest time for any one case being six months, and none have shown any signs of recurrence of aural discharge nor does the epidermis lining the radical cavity in these cases appear any different from that found in my cured cases of bone necrosis.

Following radical mastoid surgery, one of the greatest contributory factors toward poor results is the failure to produce a sufficiently adequate opening in the external auditory meatus. During the performance of the plastic operation on the posterior membranous canal wall, a resultant small meatal opening will permit the desquamated epidermis to accumulate within this cavity following complete epidermization of the cavity. The purpose of the radical operation is therefore defeated since a cholesteatomatous tumor mass will then result. Consequently, since this tumor formation will occur only if drainage is inadequate, it can be obviated by paying more particular attention to the size of the meatal opening during the performance of the plastic operation on the membranous canal.

In two of my cases such a secondary cholesteatoma resulted after radical surgery had been performed. In one instance sev-

eral radical operations were performed and a recurrence of a cholesteatoma took place each time. This case showed a complete atresia of the external canal. The operation was confined to re-opening the radical wound post-auricularly, evacuating the accumulated epithelial débris and the performance of a plastic operation on the meatus and external canal which assured a large opening. This case gave a completely dry ear within five weeks and has been under observation for six months with no sign of recurrence. The other case was similar except that the patient had had one radical operation prior to the one I performed. After following out the technique as outlined in the above case with the performance of a plastic operation on the canal to assure a wide opening, a complete cure has been obtained. This case has been under observation since for one year.

Another point which must be considered in connection with Wittmaack's theory of pneumatization is the extent to which the temporal bone may become pneumatized. We are accustomed to consider in our everyday practice that pneumatization is more or less confined to the mastoid process and the areas immediately surrounding it. We consider the finding of pneumatic cells in the zygoma, squama and occipital bones as not unusual and in the performance of our simple mastoidectomies we endeavor to eradicate down to the inner table all cellular elements which we can find. Nevertheless, on closer consideration we realize that pneumatic cells can occur in other portions of the temporal bone.[18] Since the subepithelial tissue of the tympanic cavity and antrum normally grows into the marrow spaces surrounding them, this ingrowth of subepithelial tissue may take place into the marrow spaces of the perilabyrinthine area and the petrous tip if these spaces are in direct contact with and subjected to the pneumatizing influence of the tympanic mucosa. Where such a condition occurs, there will then result pneumatic spaces in the perilabyrinthine area and in the petrous apex. Consequently, a purulency within the middle ear, which in most instances spreads into the pneumatic cells within the mastoid process, may very well spread into the pneumatic cells of the petrous apex where such exist. The pathway of infection from the middle ear in such instances may lead into the petrous tip and may result in an empyema of the petrous apex which, in its development and pathology, differs in no way from an acute coalescent mastoiditis.[19] Where an extensive pneumatization of the temporal bone exists, there-

fore, one must be on a constant lookout for an involvement of the petrous apex by a spread of the infection into it from the middle ear.

Finally, Wittmaack's teaching presents us with the following warning. Since the process of pneumatization is a continuous one from birth onward, the finding of myxomatous tissue within the mastoid process of an infant does not in any way constitute evidence of a disease of the mastoid process as a causative factor in the production of athrepsia, anhydremia or dehydration.[20] The pathological studies made in cases wherein mastoid operations have been performed for the relief of these infantile intoxications must carefully differentiate between tissue which is the seat of an acute inflammatory edema and the normal myxomatous subepithelial tissue which will be found at all times at the end of the first stage of pneumatization.

Case Reports

I. S. S., female, age 21 years. Discharging right ear since childhood. Etiology unknown.

Otoscopic Examination: Total defect in right drum. Epidermis covering inner wall. Probe inserted into attic brought down cholesteatomatous masses.

X-Ray Examination: Right mastoid process sclerotic.

Operation: November 2, 1931 — radical mastoidectomy. Cortex entered through spongy spot with a drill. Large cholesteatomatous cavity found occupying antrum area. Cholesteatomatous mass removed and matrix left intact. Posterior wall lowered. Middle ear entered. Ossicles removed. Epidermis on inner wall of middle ear and attic not disturbed. Panse plastic performed. Entire cavity packed with rubber dam and posterior wound closed.

Result: Cavity completely dry and completely epidermatized on December 4, 1931.

II. Y. F., female, age 48 years. Discharging left ear since infancy. Etiology unknown.

Otoscopic Examination: Large marginal defect in posterior portion of drum.

X-Ray Examination: Sclerotic mastoid process, right side.

Operation: December 5, 1931 — radical mastoidectomy. Cortex entered through spongy spot with aid of a drill. Antrum opened and found to be small, its walls necrotic. The diseased bone immediately surrounding the antrum removed. Posterior wall lowered and middle ear thoroughly curetted. Remains of ossicles removed. Stacke plastic performed. Cavity packed with rubber dam and posterior wound closed.

Result: Ear completely dry and completely epidermatized on January 2, 1932.

III. J. R., male, age 14 years. Intermittent right aural discharge since age of three years.

Otoscopic Examination: Large attic perforation. Foul discharge.

X-Ray Examination: Sclerotic right mastoid process.

Operation: July 14, 1931—radical mastoidectomy, right side, performed as outlined in Case I. Cholesteatomatous mass in mastoid process found. Matrix left intact.

Result: Ear completely dry and completely epidermatized on August 9, 1931.

IV. A. R. F., male, age 25 years. Right chronic otorrhea since age of ten years. Etiology unknown.

Otoscopic Examination: Large marginal defect in drum. Foul discharge.

X-Ray Examination: Sclerotic mastoid process.

Operation: September 29, 1931—radical mastoidectomy performed as outlined in Case II.

Result: December 4, 1931, ear completely dry and epidermatized.

V. N. J. B., male, age 25 years. Chronic purulent otitis media since age of ten years. Several polyps had been removed from the middle ear at various times. Middle ear curetted in 1928.

Otoscopic Examination: Large marginal defect in drum. Foul discharge.

X-Ray Examination: Sclerotic mastoid, right.

Operation: December 26, 1931—radical mastoidectomy performed as outlined in Case II. Polyps and cholesteatomatous masses removed from middle ear and antrum. Cholesteatomatous matrix left in situ.

Result: January 23, 1932, ear completely dry and epidermatized.

VI. E. B., male, age 26 years. Left chronic otorrhea since infancy. Radical mastoidectomy performed in 1926. Ear continued to discharge ever since then with no remission.

Otoscopic Examination: Marked atresia of external auditory meatus. Through small fistulous opening, polyp seen coming from old radical cavity.

Operation: November 17, 1931 — revision of radical mastoidectomy. Incision made through old scar and old radical cavity exposed. Found filled with a large cholesteatoma. All cholesteatomatous material removed and matrix left in situ. Plastic operation performed on external auditory meatus to assure a large opening. Posterior wound closed and cavity packed with rubber dam.

Result: Complete cessation of discharge and complete epidermization on December 28, 1931.

VII. E. M., female, age 29 years. Chronic otorrhea since infancy. Etiology unknown.

Otoscopic Examination: Large marginal defect in drum. Foul discharge.

X-Ray Examination: Sclerotic mastoid process, left.

Operation: November 25, 1931—radical mastoidectomy performed as outlined in Case II.

Result: Ear completely dry and completely epidermatized on January 22, 1932.

VIII. E. F., male, age 36 years. Right chronic otorrhea for ten years. Vertigo and nausea for a year and a half. Spontaneous rotary nystagmus.

Otoscopic Examination: Small marginal perforation in attic.

X-Ray Examination: Sclerotic mastoid process, right.

Operation: December 1, 1931 — radical mastoidectomy as performed in Case II.

Result: Ear completely dry and completely epidermatized on January 4, 1932.

IX. P. W., female, age 13 years. Right chronic otorrhea since age of eight years.

Otoscopic Examination: Large marginal defect in drum. Granulation tissue in attic.

X-Ray Examination: Sclerotic mastoid process, right side.

Operation: July 7, 1931—radical mastoidectomy performed as outlined in Case II.

Result: Ear completely dry and completely epidermatized on August 15, 1931.

X. E. H., male, age 44 years. Bilateral mastoidectomy at age of four years. Otorrhea followed scarlet fever. Left mastoidectomy at age of eleven years and again at age of thirteen years. One year later, radical operation performed. At age of twenty-one years, left ear re-operated. Both ears then dry. At age of thirty-two years, re-infection of left ear with persistence of discharge to date. Pressure pain over left side of head.

Otoscopic Examination: Complete atresia of left external auditory meatus. There seems to be a false membrane behind which is an accumulation of cholesteatoma. Right side shows well-performed, completely-healed radical operation.

Operation: January 15, 1932—revision of radical mastoidectomy, left side, after manner described in Case VI. Cholesteatomatous material completely removed and matrix left in situ. Plastic operation then performed on external auditory meatus to assure a wide opening.

Result: Ear completely dry and epidermatized on March 1, 1932.

XI. G. M., female, age 31 years. Since scarlet fever in infancy, had chronic left aural discharge.

Otoscopic Examination: Large defect in drum.

X-Ray Examination: Sclerotic mastoid process.

Operation: January 15, 1932 — radical mastoidectomy as outlined in Case II.

Result: April 2, 1932, ear completely dry and completely epidermatized.

XII. M. S., female, age 9 years. Right chronic otorrhea for seven years. Etiology unknown.

Otoscopic Examination: Large marginal defect in drum. Foul discharge.

X-Ray Examination: Pneumatized mastoid process with cell outlines indistinct.

Operation: February 5, 1932—radical mastoidectomy. Complete exenteration of mastoid process in which cells seemed to be filled with fibrous tissue and some gelatinous material. No pathology in mastoid process until the antrum was reached when distinct bone caries was noted. After the posterior wall was lowered the middle ear was found to be filled with granulation tissue and necrotic ossicles. Stacke plastic performed on the external canal. Cavity packed with rubber dam and posterior wound sutured.

Result: Ear completely dry and epidermatized on March 19, 1932.

Note: This case is the one referred to in the text and photomicrograph of the mastoid process is shown.

XIII. F. L., female, age 26 years. Bilateral chronic purulent otorrhea since infancy.

Otoscopic Examination: Marginal perforation in right drum, posterior portion. Left drum entirely absent.

X-Ray Examination: Both mastoids sclerotic.

Operation: April 26, 1932—bilateral radical mastoidectomy after manner outlined in Case II.

Result: Right ear completely dry and completely epidermatized on May 20, 1932. Left ear completely dry and completely epidermatized on July 14, 1932.

XIV. E. N., female, age 30 years. Left chronic aural discharge for twenty years.

Otoscopic Examination: Large marginal defect in left drum.

X-Ray Examination: Sclerotic left mastoid process.

Operation: June 20, 1932 — radical mastoidectomy performed as outlined in Case II.

Result: Ear completely dry and completely epidermatized on July 18, 1932.

XV. J. R., male, age 16 years. At age of ten years had right simple mastoidectomy performed. Has had chronic otorrhea ever since.

Otoscopic Examination: Total absence of drum and ossicles. Polyp formation in middle ear.

Operation: December 26, 1931 — radical mastoidectomy. After the incision was made through the old scar, simple mastoid cavity was exposed and found to be filled with a large cholesteatoma. Cholesteatomatous masses completely removed and radical operation performed. Middle ear completely curetted of all diseased mucous membrane, bone and polyps. Cholesteatomatous matrix left in situ. Plastic performed on external auditory canal.

Result: Ear completely dry and completely epidermatized on February 15, 1932.

XVI. S. L., male, age 52 years. Left chronic otorrhea for thirty years. Dizziness and instability for two years. Severe headaches, one year. Fistula test positive.

Otoscopic Examination: Large marginal defect in drum with polyps in middle ear.

X-Ray Examination: Sclerotic mastoid process.

Operation: December 19, 1931—radical mastoidectomy performed as outlined in Case II. Fistula found in horizontal semicircular canal. Plastic performed on external canal.

Result: Discharge still present due to a sequestrum evidently coming from the inner wall of the middle ear and antrum. Bare bone felt through the external canal. Since all labyrinthine symptoms have disappeared since the operation, no operative procedure is being undertaken to remove the sequestrum at the present time. It is hoped that it will come loose of its own accord.

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DISCUSSION

Dr. James A. Babbitt, Philadelphia: This contribution by Dr. Almour is scientific, instructive and practical. The concentrated attack upon a chronic infection in the tympanic-antrum area has been emphasized by recent efforts to substitute topical and surgical measures for the too frequently unsatisfactory results in the conventional radical mastoid procedure. This failure in result concerns not only persistence of infection, lowered audition, but also labyrinth and intra-cranial complication.

The term tympanic-antrum area was used advisedly — the conception of Morris, Cheatle and others of the middle ear cleft giving developmental rise to the three cavities, mastoid antrum, tympanum and eustachian tube, primarily continuous, and preserving that continuity, however much it may be altered in shape and modified to form three cavities, has an appropriate bearing upon the surgical field of this paper. The epitympanum and antral field in the presence of a sclerotic mastoid becomes a surgical entity and links the procedure whatever the route of entrance.

This presentation is a threefold one; a review of Wittmaack's theory of pneumatization, a new surgical adaptation to it, and conclusive findings in sixteen operated cases. From mutual interest, the privilege was accorded the writer to witness the scientific approach and skillful technique in some of the operations recorded and further comment would be superfluous.

This paper presupposes the acceptance of Wittmaack's theory of the sequence in the two forms of infantile otitis media, which result in a sclerotic or diploetic mastoid, and sometimes combination of the two. The clear cut differentiation with absence of pathology in the sclerotic type is still under authoritative controversy. There is similar difference of opinion on the etiology and relative frequency of the primary and secondary types of cholesteatoma if we accept the reports from Nager's clinic. It is important to the practical procedure of the author to substantiate the non-pathogenic sclerotic mastoid, rather than the calcium resorption, with later generalized replacement, due to capillary transmission of infection as suggested by MacMahon. This would confuse the roentgenographic picture which already presents a serious problem in accurate diagnosis of sclerosis.

A case at the University Hospital may illustrate this point. A boy of nine was admitted to the ward with history of bilateral suppurative otitis media of some years standing. From local and general symptoms, blood examination and careful X-ray study, diagnosis of active bilateral mastoiditis was made. At operation absolutely solid bony structure was found on the right side with no softening except in an almost artificially made antrum. Therefore, on the opposite side a modified attic drainage was done by canal route, with prompt recovery, smooth tympanic membrane with light reflex, and almost normal hearing. This exceptional case would have done equally well with Dr. Almour's procedure, but scarcely with the same restoration of tympanic area.

Conservative otology requires basic proof before procedure and the conclusions of such men as Aschoff, Wittmaack, Albrecht, Ruttin and

Nager, across the water — Beck, Crowe, Polvogt and others, in our own country, have prepared the field for these new suggestions. As Kopetzky tritely suggested in discussion of the earlier paper, it is sometimes necessary to retrace one's steps and follow the earlier research guidance more accurately.

In the last analysis, I am not sure a cholesteatomatous or a sclerotic etiology influences the operation selection if preliminary diagnosis is accurate. No single phase of chronic mastoid surgery has been more disquieting than the laborious and seemingly unnecessary excavating down through solid eburnation of bone in the radical mastoid. It has doubtless been this limited area of mastoid antrum finally reached, as well as the constricted tortuous line of drainage so often found in attic suppuration of long standing, that have prompted the various operative modifications offered, both by internal and external route. It has long been a query in the writer's mind whether completely adequate surgery through the auricular canal could not be devised, which would result in less destruction of annular ring and audition danger and less radical disturbance of the epitympanic ossicular contents. The Tobey modified radical would seem to have solved this, but landmarks appear doubtful and performance difficult. Dr. Almour's procedure requires precisional reaching of the antrum, sometimes difficult in infant patients, and still requires convincing argument against the popular belief that hearing is lowered.

The role of the squamous epithelium is an important one — the failure in its elimination with secondary cholesteatoma equally so. I am ready to believe that the over 50 per cent success in the modified attic drainage is primarily due to the increased epithelialization permitted. This new operative approach is a rational one, the plan of utilizing the cholesteatoma matrix in covering the cavity surface with epithelium is a distinct contribution, and the author is to be congratulated.

Dr. E. M. Josephson, New York: The too free or literal acceptance of the conclusions which Dr. Almour draws from the theories of Wittmaack is fraught with some danger. Those of us who have operated upon the mastoid will recall having often seen isolated cells in the tip and along the sinus plate, even in cases presenting the most densely sclerotic mastoid processes. Inasmuch as these cells do not always show up in X-rays of the mastoid, one cannot be sure of the presence or absence of infectious processes in those cells. Failure to drain such infectious processes is almost certain to result in a lighting up of the infection with the attendant danger of complications in mastoids traumatized by such operative procedure as Dr. Almour suggests, limited to drainage of the tympanic cavity and antrum.

The sometimes successful medical treatment of chronic mastoid infections with powders liberating deeply penetrating iodine gas, such as iodobor, and the minor operative intervention emphasized by Dr. Babbitt, questions the justification for radical mastoidectomy in all cases of otorrhœa presenting mere chronic infections of the tympanic cavity, or even in cases of chronic infection of the mastoid as a whole.

Not even complications such as the much discussed infection of the cells of the petrous tip necessarily constitute an absolute indication for operative intervention. This was brought home to me forcibly but a few days ago by the case of the son of a colleague in New York City. The child developed a Gradenigo syndrome following a mastoidectomy. The infection ran a subacute course for many weeks, and low grade meningitis was suspected. I suggested the use of Iodobor powder in the wound and the tympanic cavity, and was gratified to learn a few weeks later that the petrositis had completely subsided under the suggested treatment.

Operative intervention in chronic infections of the mastoid appears to be indicated only where modern medical therapy has proved to be of no avail after a reasonable period of trial, or in cases which at the outset show evidence of complications or danger of complications imminent. In those cases in which operative interference is indicated, however, the exposure of the field should be as wide as possible, no matter whether the mastoid be sclerotic or not, in order to insure as far as possible against overlooking any infected tissues or cells.

DR. RALPH ALMOUR, closing: I want to thank the discussers for their interest in the paper.

I am afraid that I cannot agree with Dr. Josephson as to the indications for radical surgery. I believe that when a lesion is present it should be removed from the head in order to prevent intracranial complications. In other words, I do not believe in locking the stable door after the horse is out.

As far as medical treatment of chronic suppuration is concerned, that is beside the point under discussion.

I want to say this, that it is not so much the technique of the operation; it matters very little whether one performs surgery through a postauricular incision or whether one is more competent and can do some sort of procedure by means of an ossiculectomy, — it is the principle that is important, namely, to clean out the entire middle ear, of which the tympanic antrum is a part. That can be done by some men through the external meatus; for others it is easier to do it by the postauricular route. That is immaterial; the important thing is the principle of attack.

The after-treatment in my cases consists of putting in a large piece of rubber dam, after the plastic operation is done, so as to keep the flaps in place. At the end of 24 to 48 hours the rubber dam is pulled out through the external auditory canal, and from then on nothing else but dry treatment, drying the cavity very thoroughly and instilling either iodine or boric acid powder. The shortening of the after-treatment is most gratifying.

I also want to mention that this conception of retaining the cholesteatomatous matrix and using it to line the cavity is not original with me. I understand that twenty years ago Sir John Dundas Grant advocated the same method. I make that statement so you may not be under a misconception.

CICATRICIAL STENOSIS OF THE LARYNX

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STENOSIS of the larynx may occur in the course of certain infections or may result from various forms of trauma. The chief cause, however, is improperly performed tracheotomy. In practically all of the cases of acute laryngeal stenosis requiring tracheotomy for relief of dyspnea no difficulty is encountered with decannulation after the acute process has subsided, provided a proper tracheotomy was done and adequate after-care was instituted. Failure to decannulate is most often observed in cases of high tracheotomy or faulty after-care. Among the cases of chronic stenosis of the larynx sent to Jackson for treatment it was found that in approximately one-sixth of these there had been necrosis of the cartilaginous framework of the larynx probably caused by the primary condition for which tracheotomy was done while in the remaining five-sixths, the stenosis was due either to faulty tracheotomy or to improper after-care. Observations of other writers have corroborated these findings.

In a series of twenty-eight cases of cicatricial stenosis of the larynx sent to the Bronchoscopic Clinic and treated by some form of external surgical procedure it was found that tracheotomy had been performed either through the cricoid cartilage or at a higher level in twenty-one instances. None of these cases was suitable for the more conservative methods of treatment after a low tracheotomy had been performed. In sixteen cases the airway was completely occluded by a cicatricial barrier.

The following tabulation indicates the primary conditions producing dyspnea for which tracheotomy was done and the frequency of high tracheotomy and atresia of the larynx in each group.

Primary Condition	*Number Cases*	*High Tracheotomy*	*Atresia Larynx*
Diphtheria	13	8	12
Typhoid fever	1	1	1
Influenza	1	1	0
Trauma	8	6	1
Papilloma (radium)	3	3	2
Bilateral laryngeal paralysis (post-thyroidectomy)	2	2	0
TOTAL	28	21	16

Diphtheria—The influence of intubation of laryngeal diphtheria on the incidence of cicatricial stenosis is debatable. Sewall states that in the acute inflammatory conditions associated with diphtheria laryngeal stenosis occurs where tracheotomy has been done without intubation almost as frequently as where the patient has been intubated. Properly performed intubation or tracheotomy very probably are not often followed by chronic stenosis. Faulty tracheotomy unquestionably is the most common cause of chronic stenosis. Of the thirteen cases reported as following diphtheria, twelve had atresia of the larynx. Nine cases had been intubated during the disease and later tracheotomy was done; in two, high tracheotomy had been done without preliminary intubation and in the remaining case the tube had been properly placed below the second tracheal ring. In the nonatresic case a high tracheotomy had been performed. It is interesting to note the effects of aspiration of secretion and membrane together with the prophylactic measures employed in diphtheria prevention on the incidence of intubation in laryngeal diphtheria. At the Philadelphia Hospital for Contagious Diseases* direct laryngoscopic aspiration has been practiced since 1924. The percentage of intubation cases in 1923 was 26.74. Although there was an increase in the number of intubated cases in 1924, Dr. Lucchesi stated that during the period from 1925 to 1928, inclusive, the rate dropped to 15.5 per cent (average for four years). Beginning in 1929 there was a definite increase in the number of cases intubated. He attributed this to the admission of a large number of cases of acute laryngitis caused by the streptococcus, staphylococcus and other organisms as cases of laryngeal diphtheria. The policy employed at the pres-

* Personal communication from Dr. P. F. Lucchesi, Acting Superintendent, Philadelphia Hospital for Contagious Diseases.

ent time consists of direct laryngoscopic inspection of the larynx with aspiration only of those cases presenting clinical evidences of diphtheria. Neither aspiration nor intubation is practiced in the cases of non-diphtheritic laryngitis.

Papilloma—The employment of conservative methods of treatment in papilloma of the larynx results in a normal larynx when the growths cease to recur. Direct application of radium into the larynx as was practiced in the three cases reported is not only useless in papilloma but is commonly followed by extensive perichrondritis and necrosis of the laryngeal cartilages (Clerf). In two of the cases there was an atresia of the airway with destruction of the cricoid cartilage. In one case the papilloma recurred in spite of the fact that little of the normal larynx remained.

Laryngeal Paralysis—Abductor paralysis of the vocal cords following thyroidectomy is not productive of cicatricial stenosis. Emergency tracheotomy is often resorted to, however, and when hastily performed is often improperly done. With the thyroid gland removed the trachea is more accessible in these cases and tracheotomy should be easily performed.

Trauma—Extensive injury often with a compound fracture of the larynx is a relatively common cause of cicatricial stenosis and is apparently becoming more frequent with the increasing number of automobile accidents and other injuries. Dyspnea requiring prompt tracheotomy, often improperly performed, and the occurrence of perichondritis and necrosis of cartilage in compound fractures communicating with the airway are the most common causes of chronic stenosis in this group. In four of the eight cases reported the tracheotomy tube was inserted through the cricothyroid membrane and in two the tube was introduced through an opening made in the larynx at the time of the injury. Very satisfactory results can be secured and serious stenosis avoided in cases of fracture if a low tracheotomy is performed early and proper surgical measures are employed in the treatment of the injured area (Mullen).

Diagnosis—A diagnosis of cicatricial stenosis of the larynx can usually be made without difficulty. In adults, mirror laryngoscopy may be sufficient. In children, direct laryngoscopy is necessary. Certain cases may require bronchoscopy or retrograde

laryngoscopy, through the tracheotomy opening (Tucker). Occasionally it may be difficult to determine if an airway is present. Carefully performed direct laryngoscopic examination and palpating the cicatricial barrier with a filiform bougie will often prove helpful. If the tracheotomy has been performed through the thyroid cartilage or the cricothyroid membrane the tube should be removed during the examination for in this high position it may occlude a part of the airway. Roentgen studies should always be made to rule out foreign body and also for such additional information as may be secured by this method of examination.

Treatment—Before proceeding with a plan of treatment all possible sources of infection about the upper air and food passages should be investigated and removed (Orton). General systemic studies should be carried out and syphilis should be excluded. The lungs should be carefully investigated. The presence of a pulmonary infection as bronchiectasis would contraindicate any form of surgical treatment. The form of treatment to be instituted depends on the degree of stenosis and the condition of the laryngeal cartilages. These data are secured at the time of the diagnostic study and are obtained by laryngoscopy, bronchoscopy, esophagoscopy, Roentgen studies and palpation of the neck.

In minor degrees of stenosis with little or no involvement of the supporting laryngeal framework one may consider some form of dilatation. Low tracheotomy should always be performed. Many methods of dilatation including metal plugs and rubber tubes (von Schroetter), intubation tubes (Leffert) and various modifications have been recommended. In my experience direct laryngoscopic dilatation using metal bougies as recommended by Jackson has proven satisfactory. Good results have been secured by some observers with progressive dilatation using metal dilators through the tracheotomy opening (Thost-Brüggemann). This method is of value only in selected cases.

In cases of atresia and in those cases of non-atresic stenosis that do not respond to direct laryngoscopic methods of dilatation some more radical plan of treatment is necessary. With the development of more satisfactory methods of surgical treatment I do not favor the use of intubation tubes or similar apparatus that is introduced into the larynx through its natural opening

and retained in position by mechanical means. Cases responding to these methods would usually respond to direct laryngoscopic dilatation.

The underlying principles of surgical treatment are restoration of the airway by removal of obstructing tissue and its maintenance by mechanical means during the process of cicatrization and epithelialization.

One of the greatest obstacles in the treatment of these cases lies in the fact that often there is an absence of the supporting cartilaginous framework so necessary in maintaining an open airway. Involvement of the cartilage by the primary disease or secondary infection following high tracheotomy is commonly followed by loss of normal cartilage and its replacement by firm fibrous tissue or an attempt at the formation of new cartilage. Proliferation of cartilage is most commonly seen in those cases where the posterior lamina of the cricoid is involved. This newly formed tissue consists almost entirely of a heaping up, on the posterior wall, of fibrous tissue elements containing irregularly distributed islands of cartilage with occasional areas of new bone formation. This tissue cannot be utilized in the reconstruction of the larynx but must be removed in order to secure an adequate airway. Its continued proliferation often interferes with retention of the dilating apparatus at this point. Regeneration of a normal cartilage in these cases has never been observed.

Until recently the commonly advocated method of treatment consisted of packing the newly established airway for a time, followed by the introduction of some form of dilating apparatus and a Jackson laryngostomy tube. Various modifications of tubes (Tucker) and other apparatus have been used. In extensive removal of cicatricial tissue the use of skin grafting has aided in hastening epithelialization (Arbuckle).

While excellent results were obtained in many cases by laryngostomy particularly when compared with the difficulties encountered in the earlier methods of treatment, this method nevertheless left much to be desired. Frequent changing of apparatus which was often painful, time consuming and disagreeable; the problems involved in the treatment of young children who failed to cooperate and the danger of injury of cartilage; the necessity for wearing the apparatus for months and even years all added to the problems involved in the treat-

ment of this condition. In addition there was always a question regarding the outcome in cases with extensive cartilage destruction.

These problems induced Schmieglow to devise a new method of treatment of cicatricial stenosis of the larynx. Since 1910 he had successfully employed this method and had treated eighteen cases with satisfactory results. The treatment consists of laryngo-fissure, removal of webs, strictures and obstructing scar tissue, restoring the laryngeal lumen to as nearly normal as possible and the translaryngeal fixation of a rubber tube introduced through the fissure in the larynx. He found that the tube caused little inconvenience to the patient; there was no reaction of consequence; changing of the apparatus, a serious problem in children, was unnecessary and the duration of treatment and hospitalization was shortened. In adults, where one can introduce a large rubber tube in the larynx, he dispensed with the tracheotomy tube. In children, however, he recommended that the tracheotomy tube be left in because of the danger of obstruction of the rubber tube by food particles. Age was no contraindication to the use of this method. Three of his patients were less than three years of age. The period of treatment in some of his cases was less than one month.

After using the Jackson method of laryngostomy in fifteen of the twenty-eight cases reported and noting some of the objections previously mentioned I decided to employ the translaryngeal fixation method devised by Schmieglow. Of the thirteen cases treated by his method six were under ten years of age, the youngest being two and one-half years. The method employed is essentially that advocated by Schmieglow. In the earlier cases overflow of secretion into the trachea proved troublesome and necessitated closure of the upper end of the transfixed tube by direct laryngoscopic introduction of a rubber cork. I now routinely close the upper end of the tube and all patients wear a tracheotomy cannula. They do not object to this since all wore a tube before operation and in many the larynx was atresic. It is of interest to note that a rubber tube may be left in situ for several months without a marked reaction of the laryngeal tissues or incrustation about the tube. In one case the tube was allowed to remain fifteen weeks without the development of unfavorable signs.

Several points in the operation are worthy of comment. The laryngeal or tracheal airway should be made as nearly normal as possible by removal of all obstructing tissue. The rubber tube, preferably of pure gum rubber, should be sufficiently large to fit snugly in the larynx. It should extend above and below the levels of the stenosis. If allowed to remain open at its upper end it should not extend above the upper orifice of the larynx. A nineteen gauge silver wire on a large curved cutting edge needle is used to transfix the tube, passing through the wings of the thyroid cartilage. The ends of the wire should be cut sufficiently short so that they may retract beneath the skin surface. Slight infection of the wound or at the point of entrance of the silver wire through the skin may occur but is not troublesome. Hospitalization in the case of adult patients need not be continued more than several weeks since the wound usually heals promptly.

No definite rules can be formulated with regard to the duration of the time that the rubber tube is allowed to remain in situ. In cases with no cartilage loss the tube may be removed in four to six weeks. I have removed none under six weeks and, in cases with considerable cartilage loss, have allowed it to remain for three to three and one-half months. Removal of the tube is accomplished by direct laryngoscopy using a simple form of grasping forceps. The silver wire offers no resistance to removal.

In long-standing cases of stenosis with small poorly developed larynges it may be necessary to do a second operation to introduce a larger rubber tube into the larynx. In one case that had been unsuccessfully operated elsewhere by the old method of laryngostomy an eight mm. tube only could be transfixed in the larynx. At a subsequent operation a twelve mm. tube was substituted for this.

Summary of Cases

A series of twenty-eight cases of cicatricial stenosis of the larynx found unsuitable for direct laryngoscopic dilatation is reported. All of the patients had been tracheotomized for relief of dyspnea. In twenty-one, high tracheotomy had been performed; in sixteen there was an atresia of the airway. Of the thirteen cases of post-diphtheritic stenosis, twelve were atresic.

Fifteen cases were treated by laryngostomy followed by packing the larynx and using various forms of tube dilators.

Seven of these cases were atresia and in nine a portion of the cricoid cartilage had been destroyed. One case became discouraged and discontinued treatment. Three have not been cured although an epithelialized airway is present. There is lacking sufficient cartilaginous support to maintain an adequate airway. These cases will probably be treated by the translaryngeal fixation method in combination with supporting transplants although one has already received bone transplants. Two cases have been improved and decannulated but remain dyspneic on exertion. Nine cases can be considered as cured; they have been decannulated and have ample airways and satisfactory voices.

Thirteen cases were treated by the Schmieglow method. Nine of these had atresia of the larynx; in ten there was absence of a portion of the cricoid; in three of these there was also some injury to the thyroid cartilage. Nine of these cases have been cured; one will require a second operation for the introduction of a larger tube; three are still under treatment and cannot be classified so far as end results are concerned.

Conclusions—The translaryngeal fixation operation seems to be the method of choice in the treatment of cases of cicatricial stenosis of the larynx that cannot be treated by the more conservative methods. This procedure obviates the need for frequent, time-consuming, painful dressings and changes of apparatus. The duration of treatment and the period of hospitalization are shortened. Results, even in cases where there is a loss of the normal supporting tissues of the larynx, are uniformly good.

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DISCUSSION

Dr. Henry B. Orton, Newark: I think any procedure that will shorten the time necessary to treat these cases is very welcome. Those interested in this type of a stenosis will fully realize the time and labor necessary to accomplish a successful result. Diphtheria, no doubt, more than any other disease, calls for a tracheotomy, and it would seem, therefore, that better tracheotomies would be a great factor in preventing laryngeal stenosis post-diphtheritic in character.

Tracheotomy versus intubation is an old question. Personally, I am in favor of putting the larynx at rest by tracheotomy rather than using an intubation tube. An intubation tube is a foreign body, and all foreign bodies irritate.

I had the pleasure of watching Dr. Clerf perform the operation of translaryngeal fixation by the method of Schmieglow, and was much impressed. I have performed the operation three times on different patients.

A very important point brought out by Dr. Clerf in the paper was that the procedure may be repeated, using a larger tube after a certain length of time. My cases have been about forty in number, and in some I have adhered to the old method that Dr. Clerf has mentioned; namely, laryngoscopy. Anything that will limit the period of treatment from one and a half years to six months is very welcome.

Dr. E. G. Gill, Roanoke, Va.: I wish to discuss only one aspect of Dr. Clerf's paper. He spoke of treatment as a prevention of stenosis of the larynx, and emphasized the importance of a properly performed tracheotomy. In that connection I wish to mention a procedure which I have found helpful in doing a low tracheotomy. By passing a bronchoscope an emergency tracheotomy may be performed with due deliberation, as an airway for the patient is provided, and the landmarks are more readily outlined. I mention this procedure as a method of preventing stenosis of the larynx following tracheotomy. In a series of thirty tracheotomies I have done in the past six years, I have not had a case of stenosis of the larynx.

A STUDY OF TWENTY-FOUR CASES OF NECK INFECTION

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MEDICAL HISTORY records that many a physician's desire to know more about a given disease has been aroused and stimulated by one or more experiences with patients having that disease, particularly if the outcome of some has been disastrous. This paper is the result of such experience. I believe that the subject is but indifferently understood by the rank and file of the profession, and sincerely trust that by submitting the following findings I may arouse an appreciation, the while leaning very heavily on the teachings of master thinkers, and disclaiming any originality. The text books leave much to be desired after perusal. As a result of study and experience, phenomena which were at first incomprehensible or overlooked will often become clear and easily recognized. No locality of human habitation in which there occur infections of the upper respiratory tract is exempt from these neck infections. All ages and both sexes are victims. Much suffering can be ameliorated, and many fatalities prevented. Physicians and surgeons practicing in small communities, and whose work is done in small hospitals will encounter a surprisingly large number of these cases.

Analysis of the case histories which form the basis of this paper has revealed much of which I was previously ignorant. Local symptoms and signs can be explained by the applied anatomy, and where understood and recognized indicate the site of the infection and its remedy. Fatal septicæmia can often be prevented. Thrombosis of the internal jugular vein is one of the the most fatal complications of tonsillar and pharyngeal infections. It may be associated with frank pus in the throat, or the throat infection may have been apparently slight, and may have entirely cleared up. After any throat infection, light or severe, in which a septic temperature persists, thrombosis of the internal

jugular vein should be ruled out. The management of deep pus in the neck requires, on the part of the operator, courage and decision. A knowledge of the applied anatomy of the neck makes the search for pus much surer. In spite of the fact that attention was called to this subject many years ago, cases still go on to fatal termination unrecognized. Recent cases have shown that not only the laryngologist fails to recognize this condition, but that the general surgeon is equally ignorant of it. The fact that the laryngologist is frequently called in consultation by the family physician or general surgeon makes knowledge of the subject obligatory.

The purpose of this paper is first to discuss infections of the neck, especially deep pus in the neck. The source of such infection is, as Mosher has said, "in or about the tonsils." In connection with deep pus in the neck there is often an unrecognized thrombosis of the internal jugular vein which gives a fatal septicæmia. The second purpose of the paper is to discuss the surgical route to deep pus in the neck. Mosher[1] in 1920 discussed thrombosis of the internal jugular vein from infection from the tonsils. Later[2] he discussed the surgical approach to deep pus in the neck, and outlined a definite plan of approach.

The material for this paper is based on an analysis of twenty-four cases of infection of the neck, all except two of which were treated operatively. All of the pharyngo-maxillary cases, comprising fifty-eight per cent of the total, came to me unrecognized or with a tentative diagnosis of Quinsy sore throat. The severe infections which are caused by furuncles about the face and neck are not included in this series.

Incidence of Age and Sex

There were fourteen females (fifty-eight per cent) and ten males (forty-one per cent). Among the adults and adolescents (ages fourteen to fifty-three), there was a majority of females, eight to four. Among the infants and children (ages eight months to seven years), six of each sex.

Five cases under two years of age.....20+%
Seven cases between ages of two years and seven years.....29+%
Four cases between ages of fourteen and nineteen years.....16+%
Eight cases between ages of twenty and fifty-three years.....33+%

No startling conclusions are drawn from the factors of age and sex. They may be summarized as follows:

Infancy and childhood.....50%
Adolescents and adults.....50%

Etiology

This is considered from the standpoint of the part through which the infection enters, the portal of entrance, and any known pre-existing disease or injury, contributory or provocative. The determination, by history and examination, of the exact portal of entrance should be given important consideration, for with knowledge of this it may be possible to anticipate the course of the infection when invasion of the cervical region has taken place, and gives valuable information as to the treatment, medical and surgical, best adapted to the case. For instance, if the infection of the neck results from infection of the teeth in the mandible, the submaxillary and submental regions will probably be the first part of the neck involved.

Analysis of the etiology in these cases, few though they be, definitely corroborates what Mosher has stated, which is, that the greatest single cause of these infections is "in or about the tonsils." Next in frequency comes the pharynx. Acute or subacute inflammation of the tonsils preceded or accompanied the attack in eleven cases (forty-five plus per cent). In several the tonsillitis occurred from one to two months prior to the outbreak in the neck. Lymph node inflammation was believed to be the connecting link in these. Inflammation of the pharynx alone accounts for four cases. In these the tonsils had been removed from one to three years previously. Tonsil remnants located in much deformed and scarred tonsil fossæ are to be viewed with suspicion. It may not be possible to definitely determine whether the infection enters through the tonsil or by way of the pharyngeal wall, as where there is tonsillar inflammation there is also present some pharyngeal inflammation, and vice versa. All of the mucosa of the upper respiratory tract may be inflamed, including that of the nasal accessory sinuses. The infection may begin in the nose and sinuses, and be followed by involvement of the pharynx and tonsils. Pharynx and tonsil inflammations account for fourteen cases (fifty-eight per cent). Two of these followed surgical trauma. Three cases were of dental etiology (twelve per cent).

They were all mandible in location, two following extractions, and one necessitating multiple extraction at the time of operation because of extensive alveolar abscess. The remainder (twenty-nine per cent), numbering seven cases, exhibits as many varieties of etiology as there are cases. Enumerated, these are as follows:

1. One, case No. 19, followed a tonsillectomy, the infection entering through one tonsil fossa, and remained localized.

2. One, case No. 12, followed surgical removal of an adenoid. This involved the lateral area on one side of the pharynx, the process having no resemblance to the typical retro-pharyngeal abscess.

3. One, case No. 14, was caused by infection induced by the injection of a local anesthesia along the posterior surface of the maxilla in the direction of the spheno-palatine fossa.

4. One, case No. 4, followed trauma to the mucosa of the hypo-pharynx by a bolus of food containing an unmasticated bread crust.

5. One, case No. 15, began in an infected lymph node lying in contact with the fascia of the submaxillary salivary gland. It was never possible, by history and examination, to determine the exact point of entrance of the infection invading this lymph node.

6. One, case No. 21, is regarded as having been caused by infection of the capsule of the thyroid gland. It is concluded that the infection may have begun in a lymph node lying in contact with this fascia. When first seen the case was fairly well along, and had an appearance strikingly like an ordinary goiter. There were no symptoms indicating derangement of the function of the thyroid. When first seen there was distinct fluctuation.

7. One, case No. 13, after careful consideration, is attributed to inflammation of the petrous part of the temporal bone. It could not be proven by autopsy findings because the patient got well. At the time, I talked with Dr. Wells P. Eagleton over the telephone about this case, and he advised drainage in the neck before attempting an attack on the bone. This was done, with gratifying results. The possibility that the pharyngeal infection in this case may have resulted from extension along the auditory tube from the middle ear has been considered. Radiograph of the base, vomiting, and first branch trigeminal pain point strongly to the petrous. There was at no time any paresis of the sixth

nerve. Separated by a long interval of time there had been done two mastoid operations.

It is to be noted that in this group of cases there are none caused by the following conditions: External neck injuries, disease of the cervical vertebræ, Bezold's abscess, fractures of the larynx and trachea, perforation of the æsophagus, suppurating cysts, fracture and injuries of the jaw.

The bacteriology of these cases was in no sense complete, but enough is in evidence to give basis for one conclusion, viz., that at least fifty per cent of these infections are caused by the hæmolytic streptococcus.

No culture obtained.....six cases
Organisms failed to grow.....two cases
Pure culture streptococcus hæmolyticus.....ten cases
Streptococcus, not typed.....two cases
Staphylococcus, strepto and pneumococcus, not typed.....one case
Strepto and staphylococcus, not typed.....one case
Streptococcus viridans and staphylococcus albus.....one case
Staphylococcus aureus .....one case

Applied Anatomy

A familiarity with the applied anatomy of the cervical fascia is indispensable to a comprehension of the problems arising from infection in this region. What is here said about applied anatomy is based entirely on the work of Mosher, and deserves great emphasis.

There are three large fascial compartments in the upper part of the neck which communicate more or less directly with the sheath of the great vessels. These are the parotid, the submaxillary and the pharyngomaxillary. The parotid and submaxillary compartments contain the respective salivary glands, and require no detailed description except a mention of the fact that the retromandibular process of the parotid gland is devoid of fascia at its inner extremity where it lies in the most intimate relationship with the pharyngomaxillary space. While the submaxillary gland itself is separated from the mucosa of the floor of the mouth by a muscle layer, it does get a direct communication with the mouth cavity through its duct. The pharyngo-maxillary space is somewhat cone-shaped, the apex of the cone being continuous downward into the neck as the sheath of the great vessels, and its base uppermost at the base of the skull. By using a bit of imagina-

tion these three compartments may be likened to a trifolium, the sheath of the vessels being the stem, the three spaces the leaves.

The prevertebral fascia requires special mention because of the frequency of infection. No detailed description is necessary except to mention that it encircles the vertebral column and the muscles attached to it. Infections here have a decided tendency to extend rapidly downward. I have never observed a true midline retropharyngeal abscess to invade the pharyngo-maxillary space. Perhaps this is also the explanation of the prompt recovery after incision through the mouth in cases not seen late. The applied anatomy when viewed as follows, explains these observations. See also Fig. 11.

High up in the neck, the scaleni muscles, at their origins, lie in contact with the vertebral column. Lower in the neck these muscles course forward and laterally to reach their insertions on the first and second ribs. As seen from the front there is thus formed a pyramidal space, with its apex up at the skull base, its posterior side formed by the vertebral column and longus colli muscles, and the lateral sides by the scaleni muscles. Infections starting high up in the median line therefor would have a natural tendency to extend downward along the longus colli fascia rather than in a lateral direction. Infections which are lateral as well as midline at the start could extend laterally as well as downward, and quickly reach the vascular sheath which crosses the scalenus anticus obliquely.

A true midline infection would therefor extend into the posterior mediastinum along the vertebral column all the way. The lateral infection would travel forward along the scaleni muscles under the clavicle and into the costo-caracoid region of the chest wall, into the axilla, and also possibly into the posterior cervical triangle, where it would become superficial. It can be assumed therefor that if the infection takes this lateral course it has been in a lateral situation from the start, and should be differentiated from a true midline retropharyngeal abscess.

One other layer of fascia is of importance from the standpoint of applied anatomy. It has been given various names according to locality. This fascia encircles the visceral space of Tillaux,[3] and is therefor well named the visceral layer. In the lower part of the neck it is also known as the pretracheal layer, and surrounds the trachea and æsophagus. It splits anteriorly

to envelop the thyroid gland. In the upper part of the neck it is known as the bucco-pharyngeal fascia, and covers the constrictor muscles of the pharynx and the buccinator. This is the fascia which is the seat of involvement when infections penetrate from the hypopharynx and æsophagus. I earnestly recommend the reading of Mosher's description of the applied anatomy of the cervical fascia as published in the Transactions of this Society for the year 1929.

Slides

Figure No. 1 shows the pharyngomaxillary fossa as seen from behind. The superior constrictor of the pharynx forms its inner wall, the internal pterygoid muscle and the retromandibular process of the parotid gland its outer wall. The structures attached to the styloid process cross it, dividing it unequally.

Figure No. 2 shows the close relationship of the parotid and the submaxillary glands to each other and to the great vessels.

Figure No. 3 shows the point at and under the angle of the jaw where the layer of fascia on the deep surface of the two salivary glands thickens to form the stylomandibular ligament. This is the landmark which is felt with the finger in the approach to all three of these spaces.

Figure No. 4 shows the manner in which the internal pterygoid forms the outer wall of the space. It also shows the stylomandibular ligament.

Figure No. 5 is a diagrammatic cross-section drawing of the pharyngomaxillary space and the structures bounding it on two sides. It explains three important signs of infection in this space, — swelling of the lateral pharyngeal wall by displacing the superior constrictor into the pharynx, trismus due to splinting of the internal pterygoid, and swelling of the parotid gland region by extension of the inflammation into the sheath of the gland.

Figure No. 6 shows a skeletonized drawing of the buccopharyngeal and visceral fascia, and explains how an infection may extend from the skull base to the chest. It also shows how an infection may encircle the trachea and æsophagus.

Figure No. 7 is a skeletonized diagrammatic drawing showing the location of some of the fascial compartments in the neck, and their relationships.

Figure No. 8 is the same with the head omitted.

Figure No. 9 is the same somewhat altered, but showing the same relationships.

Figure No. 10 is the same altered to represent a trifolium.

Figure No. 11 shows the bony and muscular framework upon which the prevertebral fascia is reflected. When thus viewed from the front it has a similarity to the appearance of the inside of a tent when viewed through its open door. It explains why a true midline infection tends to remain so.

Figure No. 11-A shows a normal internal jugular vein from a newborn infant which died of congenital heart disease.

Figure No. 12 shows a section through internal jugular vein, Case No. 12. *A*, Polynuclear leucocytes. *B*, Erosion of endothelium. High power.

Figure No. 13—Another field from the same slide.

Figure No. 14 the same under oil immersion.

Figure No. 15 shows a section through lymph node dissected off the jugular sheath, Case No. 12, showing (*a*) polynuclear leucocytes near the capsule.

Figure No. 16 shows a cross-section through the whole of one side of a neck showing very extensive inflammatory involvement of the fascial planes and jugular vein which was thrombosed. All this without visible external swelling of the neck. Symptoms in this case were predominantly respiratory in character and the patient died after tracheotomy. It is obvious that there was no obstructive swelling of the tracheal membrane. For the use of this slide I am greatly indebted to Dr. Harris P. Mosher, whose case this was, and I also wish to thank Dr. D. K. Judd of the Massachusetts Eye and Ear Interne Staff for the lantern slide and photograph.

Figure No. 17 shows the same section under higher power, showing the inflammatory infiltration in and around the internal jugular vein.

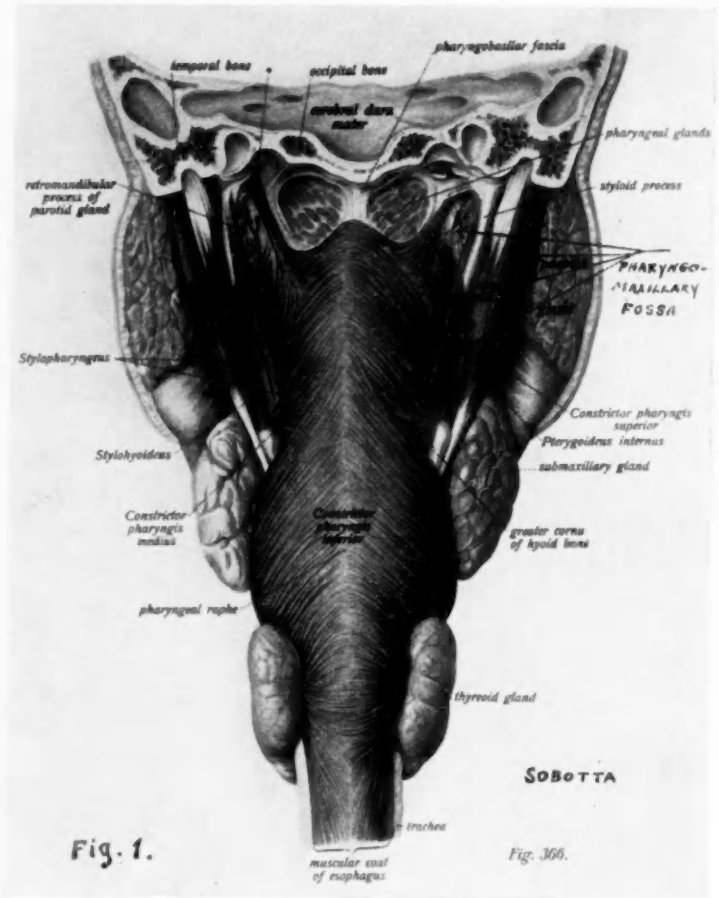

FIG. 1—Pharyngomaxillary fossa from behind.

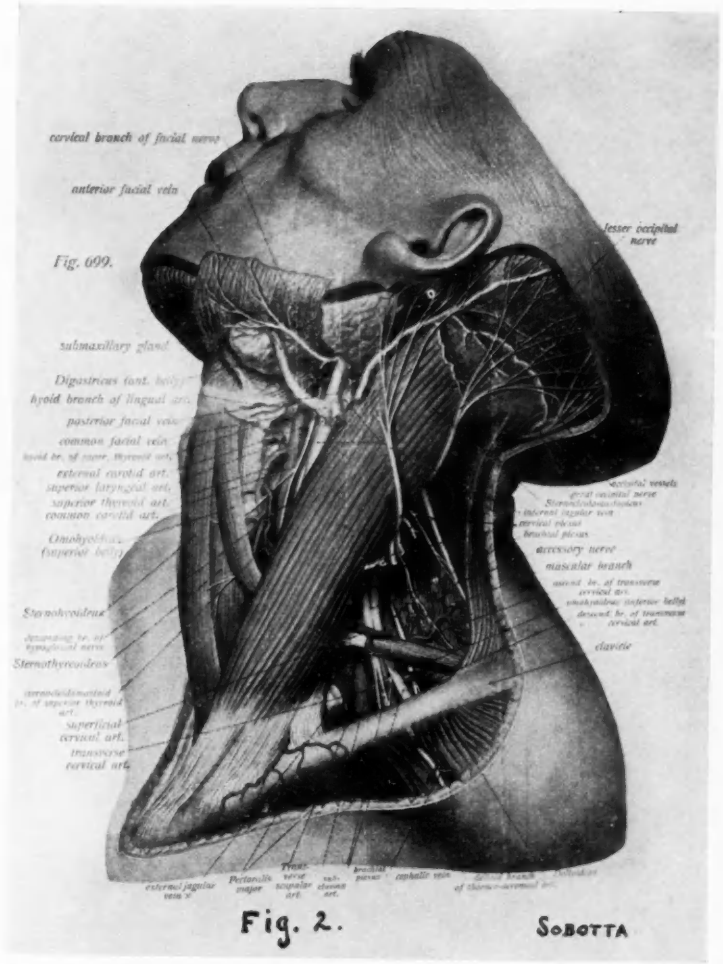

FIG. 2—Close relationship of the parotid and submaxillary glands to each other and to the carotid sheath.

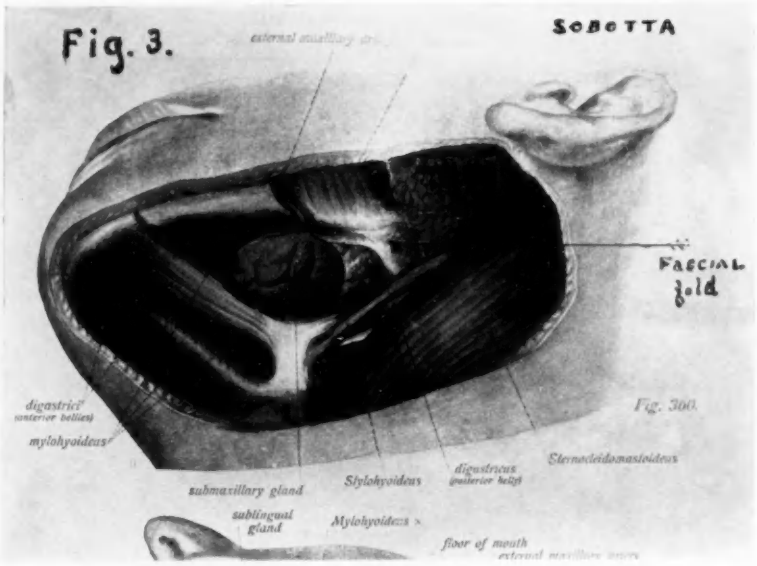

FIG. 3—Fold of fascia between the parotid and submaxillary glands.

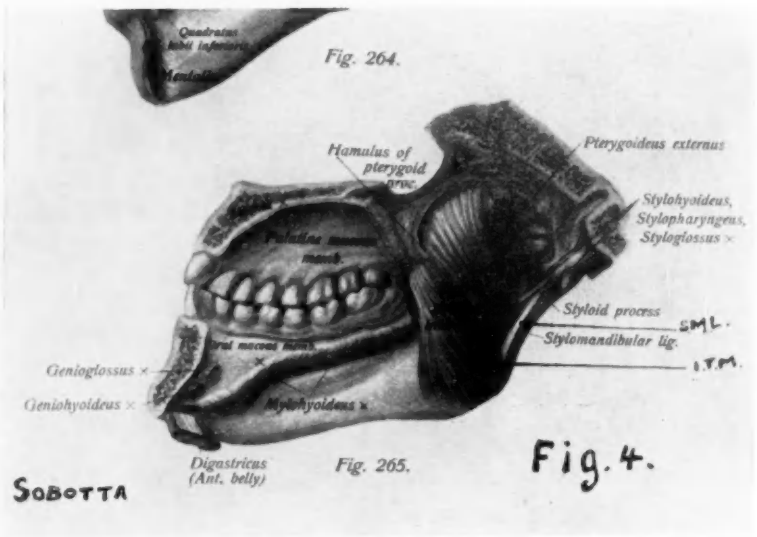

FIG. 4—Stylomandibular ligament and internal pterygoid muscle.

Fig. 5.

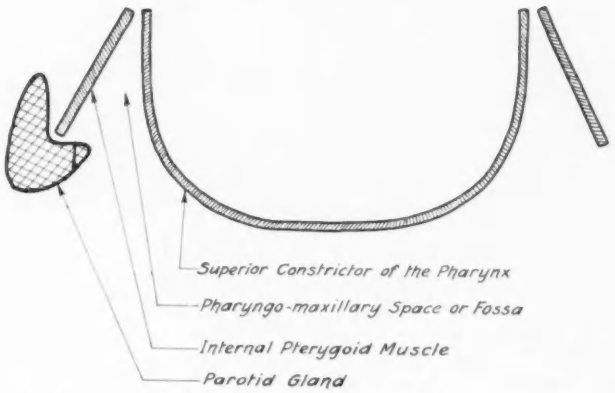

FIG. 5—Diagrammatic cross-section drawing of the pharyngomaxillary fossa.

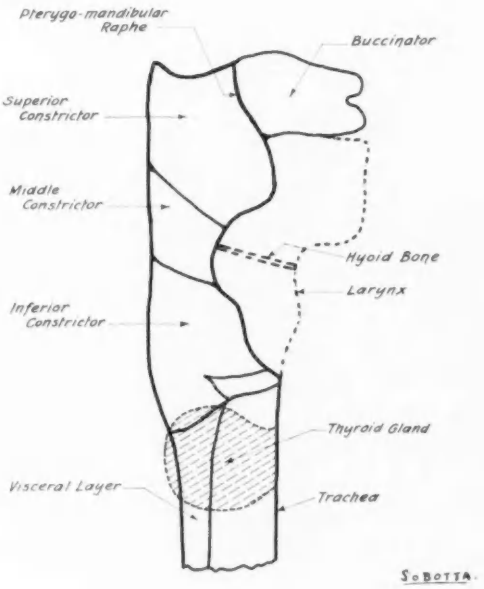

Fig. 6.

FIG. 6—Skeletonized drawing of the buccopharyngeal and visceral fascia.

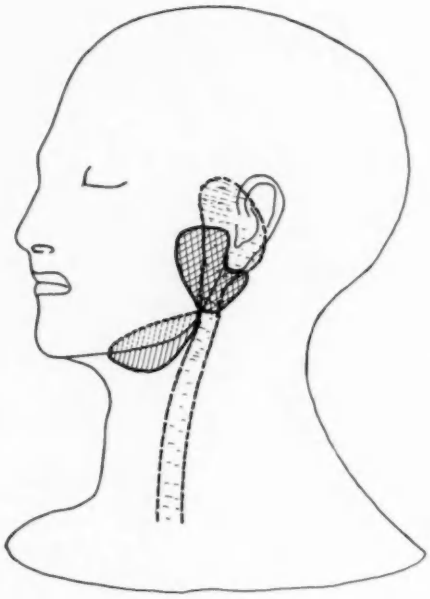

Fig. 7.

FIG. 7—Shows location of fascial compartments in upper part of neck.

Fig. 8.

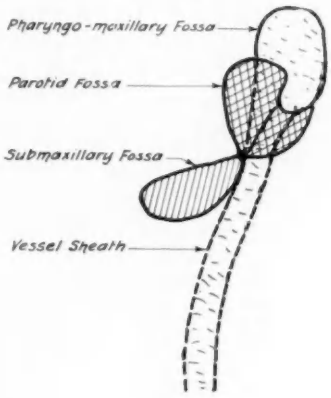

Fig. 9.

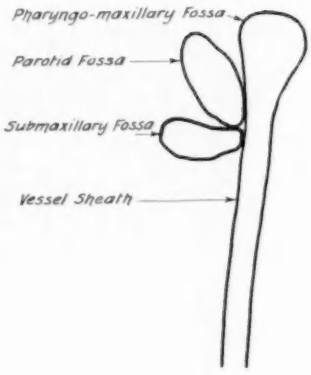

Fig. 10.

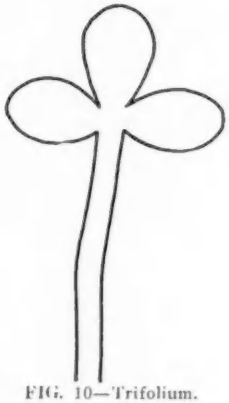

FIG. 10—Trifolium.

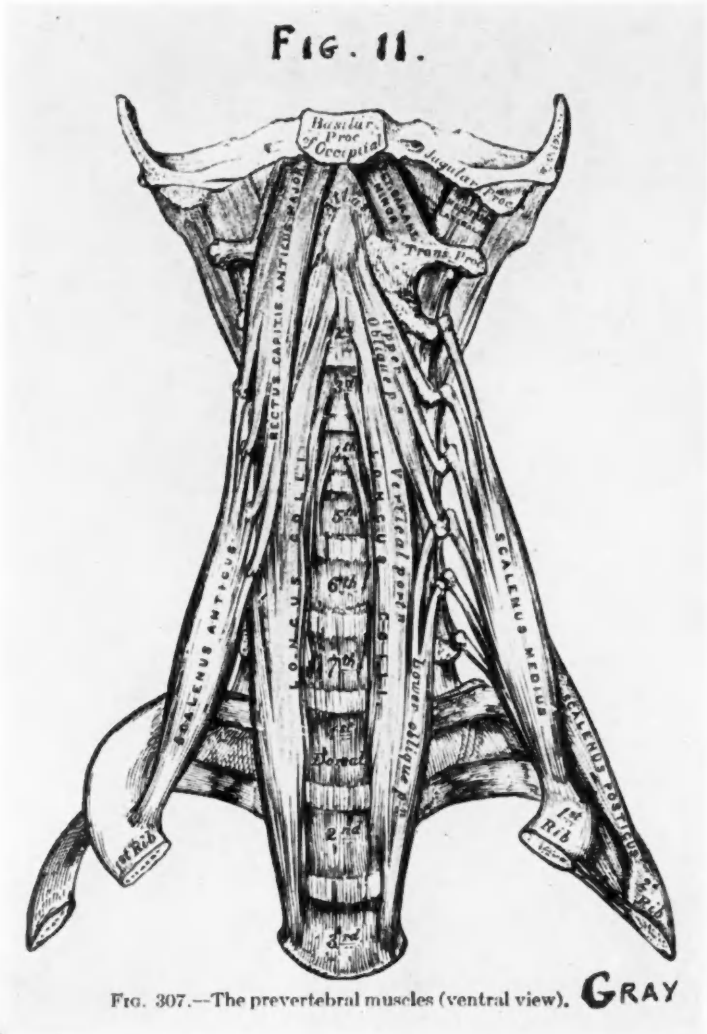

FIG. 11—Shows the bony and muscular framework upon which the prevertebral fascia is reflected. When thus viewed from the front it has a similarity to the appearance of the inside of a tent viewed through its open door.

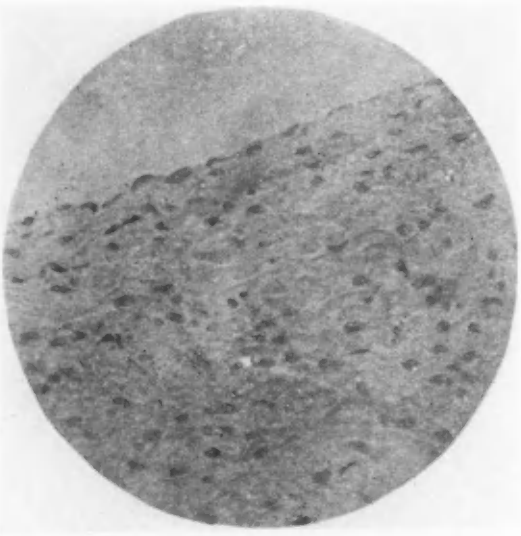

FIG. 11-A—Normal internal jugular vein.

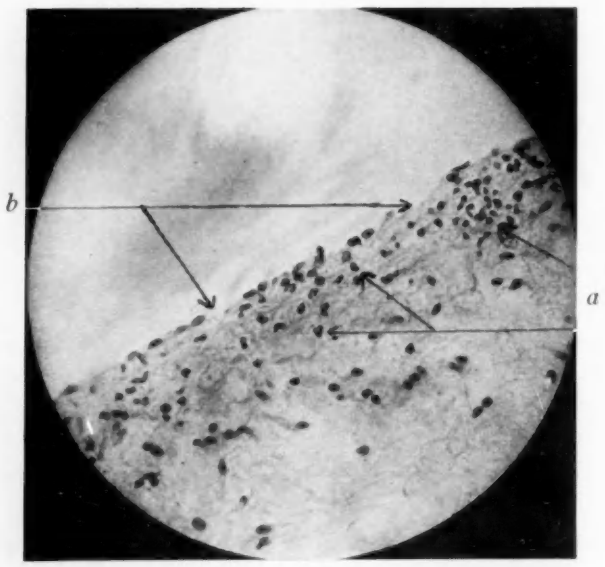

FIG. 12—Section through internal jugular vein, Case No. 12. *A*, Polynuclear leucocytes. *B*, Erosion of endothelium. High power.

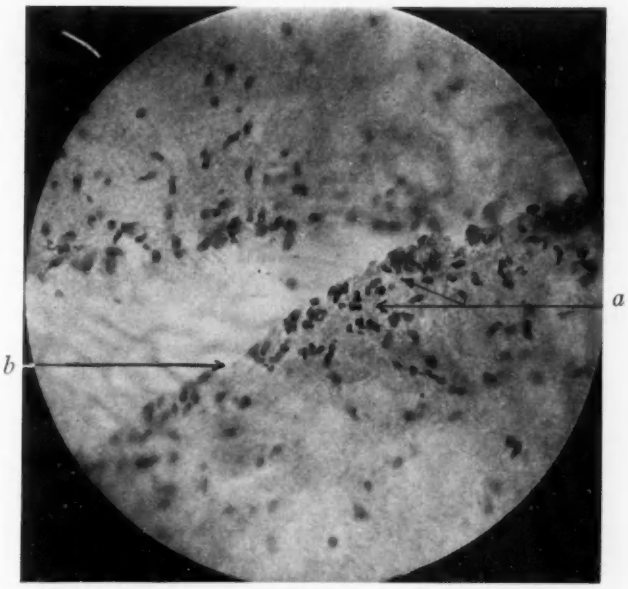

FIG. 13—Section through internal jugular vein, Case No. 12. (*a*) Polynuclear leucocytes. (*b*) Erosion of endothelium. High power.

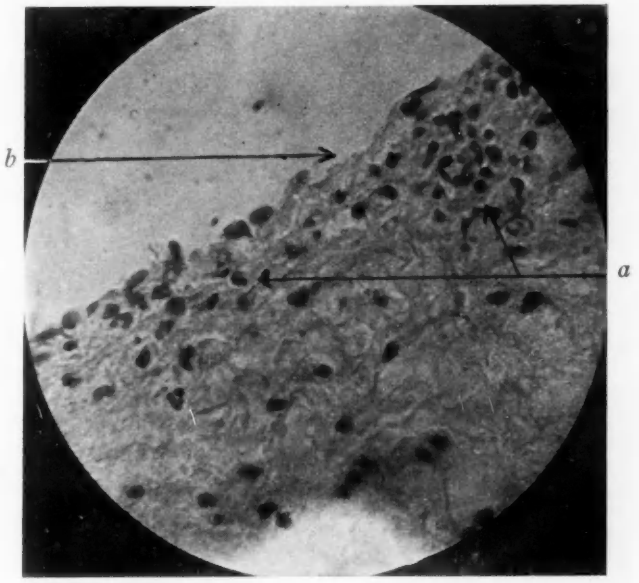

FIG. 14—Same as Figure 12, with oil immersion.

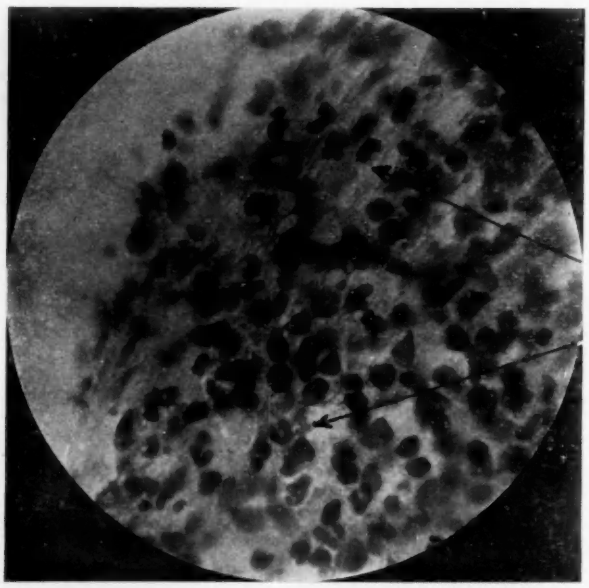

FIG. 15—Section through lymph node dissected off the jugular sheath, Case No. 12, showing (*a*) polynuclear leucocytes near the capsule.

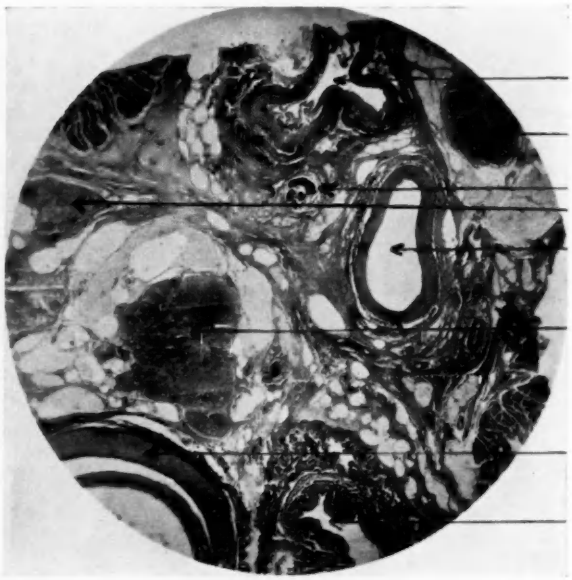

FIG. 16—Cross-section through the whole of one side of the neck showing very extensive inflammatory involvement of the fascial planes and jugular vein. All this without visible external swelling of the neck. For the use of this slide I am greatly indebted to Dr. Harris P. Mosher, whose case this was. (Low power). (*a*) Internal jugular vein. (*b*) Vagus nerve. (*c*) Common carotid artery. (*d*) Thymus gland. (*e*) Trachea. (*f*) Æsophagus. (*g*) Lymph gland. (*h*) Lymph gland.

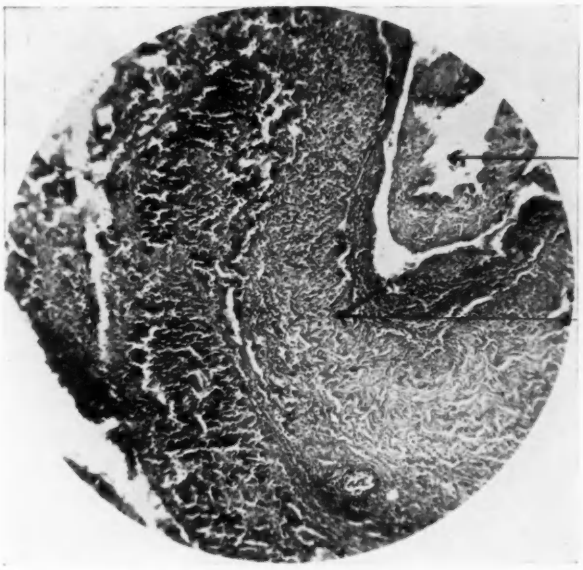

FIG. 17—Same section under high power showing the inflammatory infiltration in and around the internal jugular vein. (*a*) Internal jugular vein. (*b*) Perivascular sheath.

Regional Manifestations

The following grouping of fascial plane involvement is an attempt at classification, and is suggested by the findings. It should be obvious that in these severe inflammatory conditions the involvement does not remain strictly confined to one particular fascial compartment.

1. Pharyngo-maxillary space infection.....14 times—58 %
2. Submaxillary space infection..... 5 times—20+%
3. Carotid sheath infection..... 5 times—20+%
4. Cervical lymph node suppuration..... 3 times—12+%
5. Prevertebral fascia infection..... 3 times—12+%
6. Buccopharyngeal, pretracheal or visceral fascia infection 2 times— 8+%

Pharyngomaxillary Space Infection

From this summarized list it will be seen that infection in the pharyngomaxillary space is frequent, over half of the cases. Even in those where the beginning of the infection is distant from this space, as where the primary manifestation is in or near

the submaxillary space, there will sometimes occur extension of the infection into the pharyngo-maxillary space. In fact it should be anticipated, and daily inspections of the pharyngeal wall made. Involvement should be considered present if there is trismus and swelling of the lateral pharyngeal wall with or without change of color. With the swelling of the lateral pharyngeal wall there is often what I have chosen to designate as a prolapse of the tonsil and palatal arch, which is in reality a displacement toward the median line of the mouth. This prolapse or displacement can and does occur without the slightest swelling or enlargement of the tonsil itself, and helps to differentiate it from a peritonsillar abscess in which inflammatory swelling of the tonsil is present. When this fullness of one side of the palate is present the uvula has been seen to point almost horizontally toward the opposite side. In some cases there is very little, if any, inflammatory reaction to be seen with the swelling, but in other cases it is present. If present it must be differentiated from peritonsillar abscess, and this is not always easy. Swelling of the lateral pharyngeal wall is an aid in the differentiation, but is sometimes hidden by the palatal or faucial swelling. If there is swelling over the region of the parotid gland it may be considered additional evidence of deep infection. Where the infection is lower in the neck below the level of the angle of the jaw it does not involve this space, and these signs would not be present. When the pharyngomaxillary space is the seat of the initial invasion in the cervical fascia, that is to say, not the result of extension from one of the other compartments of the fascia the etiology is most frequently infection in the tonsils and pharynx. Infection in the nose, sinuses, adenoid, pharyngeal lymph nodes, cervical vertebræ, mastoid and petrous temporal bone are all possible primary sources.

Inflammations in the parotid and submaxillary spaces not rarely extend into the pharyngomaxillary space. There may be infection in the pharyngomaxillary space without any visible evidence of infection in the parotid or submaxillary spaces. Inability to open the mouth widely is one of the earliest signs of infection high up. Infection severe enough to cause phlebitis of the jugular vein early may exist without visible external swelling of the neck. If not drained such cases will go on to jugular thrombosis, and die of a fatal septicæmia. Infection outside of the

tonsil remains a peritonsillar abscess so long as it does not penetrate the muscle bed forming the tonsil fossa. As soon as the infection penetrates the pharyngeal constrictor muscle it becomes pharyngomaxillary. This penetration may take place directly or through the veins as was beautifully shown by Waldapfel[4] at the meeting of this Society in 1928.

Submaxillary Fascia Infection

This group, comprising the so-called cases of Ludwig's Angina, may be subdivided into two groups, which, however, manifest themselves similarly. One group is of dental origin, and the other is not. The cause of this latter group is sometimes obscure. In two of the cases it was attributed to lymph node inflammation. Though there were none in this series it is to be expected that infection in the floor of the mouth may be considered as a starting point. It is of significance, however, that the only cases in this series which had swelling and induration of the floor of the mouth were of dental origin. The inflammation rarely remains confined to the submaxillary space, but extends into the parotid space or into the pharyngomaxillary space or both, and downward below these into the lower part of the neck along the vascular sheath. The applied anatomy of the fascia of the submaxillary triangle region suggests that infection would find a barrier to direct downward extension at the hyoid bone where the fascia gets a firm attachment. For this reason deflection laterally to the vascular sheath is to be expected if extension is not checked. This observation is borne out clinically. It is noteworthy how frequently swelling in the submaxillary space becomes the initial manifestation of infection in the neck. Those of dental etiology yield promptly to submaxillary drainage, and can usually be given a good prognosis. In these trismus is extreme, and the floor of the mouth is usually involved. Those not of dental etiology are an insidious and dangerous type, and there being no primary involvement high up, swelling of the lateral pharyngeal wall of the naso and oro-pharynx and trismus would not be present. If later there is extension into the pharyngomaxillary space these signs will appear. These cases should be drained early, and if sepsis continues and chills occur the carotid sheath should be immediately exposed. If blood cultures are positive ligation or resection cannot be too quickly done, for

thrombophlebitis in distant veins may occur early. If sepsis characterized by wide fluctuations of temperature persists with negative blood cultures, ligation or resection is nevertheless indicated. This is particularly so if there are chills. All of these manifestations have occurred in cases in this series.

Carotid Sheath Infection

This, as can be explained by the applied anatomy, is secondary to infection of any or all of the other compartments and their contained lymph nodes. When the infection begins in the submaxillary or parotid compartments it may extend directly to the sheath of the vessels. It may, however, extend to the pharyngomaxillary space first, the infection of the sheath becoming a later development. A tympano-mastoid infection may intervene, and in turn cause a sigmoid sinus thrombosis. Tenderness on palpation over the carotid sheath and deep to the sternomastoid muscle is an unreliable sign of vein infection; so also is swelling; for both may be caused by inflamed lymph nodes, and not necessarily by inflammation of the vein wall. One should be guided more by the degree of sepsis than by these local signs, and absence of local signs should not be construed as sufficient reason to rule out the possibility of jugular sheath infection. Likewise their presence cannot be made the sole reason for jugular surgery.

There were five cases of carotid sheath infection, two of which did not go on to thrombosis. One of these, case No. 15, disclosed a peculiar fibrinous exudate on the sheath when it was exposed, and because I did not at that time realize the significance, the vein was not resected. After a stormy course, and aided by blood transfusions, the patient recovered. The other, case No. 12, is the one from which the photomicrograph was made, which shows the polynuclear infiltration of the vein wall. Blood cultures were negative in both of these. In one of the three cases in which there was thrombosis, case No. 16, the thrombosis occurred in the sigmoid sinus and was caused by a coincident tympanomastoid infection. In this the blood culture was positive. In the other two cases, the thrombosis was caused directly by the neck infection. One of these, case No. 8, was one of my earliest cases, and was draining profusely through an incision in the lateral pharyngeal wall. Because of this and the

marked subsidence of the swelling of the neck, it was concluded at consultation with general surgeon and another laryngologist that external drainage was not necessary. The patient died of sepsis caused by jugular vein and cavernous sinus thrombosis. In the other, case No. 24, the carotid sheath infection was secondary to the submaxillary and parotid space infection, and after obstructive thrombosis had taken place the blood culture which had been positive become sterile. It is urged therefor that blood cultures be made early in the course of the disease because of the possibility that negative results will be obtained after the vein is corked off by the thrombus. The patient who died, case No. 8, gave negative blood cultures, but they were done late in the disease, and the negative results can now be explained on this basis. Blood cultures are most apt to be positive if the blood is taken during or immediately after a chill.

It cannot be too strongly emphasized that phlebitis of the jugular vein can take place early. Metastatic abscesses are to be expected when thrombosis occurs.

Cervical Gland Abscesses

The superficial glands which lie between the superficial fascia and the superficial layer of the deep fascia, drain into the glands of the deep fascia. Suppuration of localized groups of lymph nodes is common. This can occur without demonstrable involvement of the fascial planes, other than that which is present at the place where the glands are suppurating. In other words, it is then strictly localized, and in fact the suppuration brings about localization. In such cases incision should be delayed until fluctuation or inflammation of the skin is present, provided that there is no sepsis and no evidence of deep involvement. There is another type of case in which the individual glands on the deep fascia cannot be felt, and there is no localized suppuration. There may be only a slight fullness along the sternomastoid muscle with well marked tenderness on pressure or manipulation of the muscle due to inflammation of the nodes beneath it which lie on the carotid sheath. In these, suppurative disintegration of the glands takes place late or not at all, and sepsis and blood culture examinations become the important indicators. Spontaneous recovery is possible and common, but an occasional case will tax the best surgical judgment. In two cases in which

large superficial abscesses were evacuated the inflammation did not remain confined to the glands though free drainage had been established, and there took place extension into compartments of the deep fascia. This changed the picture, and necessitated the introduction of drains into the fascial compartments invaded. In one of these, case No. 15, the infection lay dormant in a lymph node in contact with the submaxillary salivary gland for several months, when suddenly it became acutely inflamed, involving the salivary gland compartment, and spread to the pharyngomaxillary and parotid compartments. Before the inflammation was under control, it had invaded the carotid sheath.

In the other, case No. 10, the infection began in the submaxillary and upper cervical lymph nodes, rapidly formed an abscess which was promptly incised and drained. Everything progressed as though it were going on to a prompt recovery in a few days, when suddenly the infection extended along the fascia, invading the parotid, submaxillary and pharyngomaxillary compartments, and the sheath of the vessels high up. It is to be concluded therefor that if the infection remains superficial, and confined to the lymphatic elements, simple drainage incision through the skin and superficial fascia into the abscess cavity will bring about prompt recovery. If, however, the deep fascia is involved extension is to be expected, and the compartments invaded must be drained. Adherent, inflamed lymph nodes on the jugular sheath make ligation and resection a slow and tedious procedure. In the case No. 12, from which the photomicrograph was made, showing polynuclear infiltration of the vein wall, there were adherent, enlarged and inflamed nodes on the sheath which were carefully dissected off. Under the microscope these also showed polynuclear infiltration, but no visible abscesses. It would seem entirely reasonable to conclude that infection of the vein wall may be caused by infected lymph nodes. In this type of case there should be no hesitation to divide the belly of the omohyoid muscle if it interferes with proper exposure.

Prevertebral Fascia Infection

This group constitutes the well-known retropharyngeal abscess as seen in infants and children. There were three cases in this series, the ages of which were eight months, nine months, and six years. An abscess in this locality tends to extend down-

ward, so that when it reaches the hypopharynx, the swelling protruding forward, caps the larynx, and seriously embarrasses respiration. If in addition it extends laterally it may endanger the pharyngomaxillary space high up, or the carotid sheath lower down. I have never seen lateral extension of a typical midline retropharyngeal abscess.

I have observed one very unusual case, not included in this series because it was never proven to be inflammation of the cervical fascia. This case, after considerable study with orthopedic surgeons and internists, was attributed to myositis, possibly hæmorrhagic, involving the muscles attached to the cervical spine, occiput and mastoid, viz., the trapezius, recti capiti, obliqui capiti, splenius and complexus. The patient made a spontaneous recovery. My reason for mentioning the case is that infection of the prevertebral fascia had to be ruled out, and to call attention to the character of the torticollis. The muscles mentioned were splinted in contracture, causing the chin to be drawn toward the same side with elevation of the shoulder. This is the opposite of what takes place when the sterno-mastoid muscle is splinted in contracture by infection beneath it.

When a patient with the typical retropharyngeal abscess is seen before respiratory embarrassment has occurred, simple peroral incision is usually promptly effective. If not, Dean's[5] external route for drainage is an excellent and easy method of approach. Where there is already severe dyspnea from respiratory obstruction it may be necessary to first relieve this by tracheotomy, particularly if there is considerable exhaustion. It may not be possible to make an exact diagnosis before this is done. Years ago one such case fell to my lot. It is reasonable to suppose that if neglected, these otherwise simple cases of retropharyngeal abscess die of asphyxia or pneumonia following rupture into the air passages, rather than by extension into the chest.

Buccopharyngeal, Visceral or Pretracheal Fascia Infection

This is one of the most important fascial layers in the neck, and is given emphasis because of its contained viscera. In this series there were two cases, one of which, Case No. 4, was mentioned under etiology as having been caused by the trauma

induced by an unmasticated bread crust. Infections beginning in the hypopharynx, which penetrate the visceral walls involve this layer. Swelling is apt to appear in the hyoid and laryngeal regions laterally. When the infection is high up, lateral to the naso- and oro-pharynx, this becomes a pharyngomaxillary space infection.

This is the type of case which may sometimes be drained by internal incision, working through the laryngeal or æsophageal speculum. In doing this in the region of the pyriform sinus it is important to avoid cutting the internal branch of the superior laryngeal nerve. Dysphagia, dyspnea and hoarseness are to be expected. If internal drainage does not check its progress, external drainage should be done, as downward extension is a possibility. In the bread crust case there occurred an abrasion of the right wall of the hypopharynx near its junction with the æsophagus. An abscess developed at this point in the wall accompanied by moderate diffuse swelling of the neck over the region lateral to the larynx. It was drained by per-oral incision through a laryngeal speculum, and the patient made a complete, but slow, recovery. For a number of days an external incision was under consideration, but was not done because drainage was good, and the patient was not septic nor dyspneic. This type of case should be closely watched for downward extension, for involvement of the vessel sheath, and for laryngeal and tracheal obstruction.

There was one other case, No. 21, classified under infection of the visceral fascia. The patient had received much conflicting medical advice, which, coupled with a natural desire to avoid surgery, brought her to the operating room late. The swelling and tenderness began in the region of the thyroid gland, and being a negress, redness of the skin could not be easily seen, though fluctuation was present. A week or more elapsed before the patient could be convinced that there was pus in the neck. It is interesting to note that while there was great increase in the degree of neck swelling on both sides there was at no time downward extension dangerously close to the clavicle. When it was finally incised over the posterior border of the sternomastoid muscle all four fingers of the hand could be passed between the æsophagus and the vertebral column to the opposite side of the neck. A very large quantity of pus was evacuated.

At no time was there dyspnea, hoarseness, aphonia or dysphagia. In other words, no visceral compression nor functional embarrassment of the recurrent laryngeal nerves was present. After complete healing had taken place, and the patient discharged, the opposite side of the neck began to swell, this time being characterized by marked induration. Localization was slow, and when the lower part of the posterior triangle became involved incisions, one high and one low, were made, resulting in gradual recovery. Considerable cellulitis existed this time in contrast with the suppurative softening found previously on the other side. Downward extension into the chest would no doubt have been fatal. I am under the impression that this complication is rare except in cases of æsophageal trauma and perforation.

Surgical Approaches for Drainage

The actual line of incision through the skin is of secondary importance providing it is made long enough to give a wide exposure. Mosher, who first definitely outlined the approach to the deep fascial spaces of the neck, wisely advises a generous T-shaped incision which gives a wide, flat exposure. Through this, veins, which are apt to flood the field with blood, are more easily seen and tied. The cross-bar of the T runs parallel with the border of the jaw and close to it. Its object is to expose the submaxillary salivary gland. After the facial vein is tied and cut the lower border of the gland is elevated near its lateral extremity, and the finger inserted beneath it and carried backward and upward along the deep layer of its fascia until the stylomandibular ligament is felt beneath the angle of the jaw. It is then in position to enter any of the three uppermost deep fascial spaces. This is a very easily felt band of fascia, and after being located, the finger is carried upward along it until the styloid process is felt. By carrying the finger upward and external to the styloid process to the skull base it has entered the pharyngomaxillary space. By carrying the finger forward from the ligament along the inner side of the jaw it gets into relationship with the side of the tongue, floor of the mouth and the region of the tonsil. One of the fingers of the other hand may be inserted into mouth and pharynx as a guide, being careful to observe aseptic technique by changing the glove when withdrawn. The vessel sheath lies deep to the styloid process, and is, therefor, also drained at that point by this maneuver.

If the infection is mostly deep and more or less limited to the pharyngomaxillary fossa the dissection exposing the submaxillary gland is comparatively easy because the tissues superficial to the gland are not inflamed and swollen. In most of my cases the submaxillary region was inflamed and considerably swollen so that it was more difficult to recognize structures. In these I have used a single long incision running parallel to the border of the jaw and extending nearly to the mastoid tip. The deep fascia is exposed, carefully snapping and tying all vessels. At a point directly behind the angle of the jaw a closed blunt artery clamp is thrust through the deep fascia, and the opening enlarged sufficiently to give room for the introduction of a finger. Passing the finger beneath the angle of the jaw the stylomandibular ligament is felt for, and by the maneuver above described enters the desired fascial space. If it is desired to expose the vessel sheath I prefer to work through a longitudinal incision having its upper end at or near the angle of the jaw, and running along the anterior border of the sternomastoid muscle. At the upper extremity of this incision one may carry out the necessary finger maneuvers to enter the fascial spaces after locating the stylomandibular ligament. Inflamed, swollen and indurated tissues are concededly more difficult to recognize than those not thus involved, and a cadaver dissection is not always feasible nor desirable. With a few simple rules such as are here outlined this detailed recognition is not necessary. When, however, it is necessary to drain the vessel sheath or resect the jugular vein, anything short of complete exposure is unthinkable.

Dean[6] has recommended that all retropharyngeal abscesses be drained by the external route, incising along the anterior border of the sternomastoid muscle lateral to the larynx, reaching the visceral fascia, and following this around to the prevertebral fascia, thrusting the finger or a blunt instrument through into the abscess cavity. In cases where there is mechanical respiratory obstruction this method would avoid preliminary tracheotomy. As he states, it can be also reached through an incision along the posterior border of the muscle, but the anterior incision minimizes manipulation of the carotid sheath, and avoids the underlying sympathetic nerve.

The following is a brief group summary of the surgical approaches.

1. Prevertebral fascia infection or retropharyngeal abscess.
   (a) Direct incision of the pharyngeal wall through the mouth.
   (b) Incision along anterior border of sternomastoid muscle lateral to the larynx as recommended by Dean.
2. Pharyngomaxillary, parotid and submaxillary space infection.
   (a) Approach through submaxillary space after elevating the gland as recommended by Mosher.
   (b) Approach beneath angle of jaw directly without elevating the gland.
3. Carotid sheath and visceral fascia infection.
   Incision along anterior border of sternomastoid muscle as for jugular resection.
4. Posterior triangle infection.
   Incision along or behind the posterior border of sternomastoid muscle.

Complications

When mentioned as they occurred in individual cases their multiplicity is shown, and also the combinations in which they appeared.

Rupture through the wall of the external auditory canal took place in one patient, Case No. 7, in whom there was severe suppuration in the parotid sheath. Such a condition demands careful examination to differentiate it from an acute otitis media. Both conditions may be present at the same time.

Thrombosis of the internal jugular vein in one patient, Case No. 24, was the direct result of extension from the submaxillary and parotid spaces, there having been at no time involvement of the pharyngomaxillary space. Because of the subsidence of the neck swelling and the development of abdominal distension it was the opinion of a number of consultants that the cause of the sepsis was in doubt, and exposure of the vascular sheath and resection of the internal jugular vein was delayed twelve days. At this time the vascular sheath was exposed, and the vein found completely thrombosed and so much macerated that ligation or resection was impossible. Following this there occurred thrombophlebitis of the veins of both legs with subsequent abscess formations requiring drainage. There also occurred the

same pathology in one side of the pelvis necessitating drainage. Blood cultures showed streptococcus hæmolyticus. Cystitis and choked discs added to the patient's misery. At the time of writing this patient is still in the hospital, a sojourn of three months.

Thrombosis of the internal jugular vein and cavernous sinus occurred in one patient, Case No. 8, and caused the only death in the series.

Acute mastoiditis with sigmoid sinus thrombosis occurred as a complication in one patient, Case No. 16, and was followed later by the development of an abscess in the hip joint which required drainage. In this patient there was also homolateral paralysis of the tongue and of the pupillary fibers of the cervical sympathetic nerve. Blood culture showed streptococcus hæmolyticus.

Phlebitis of the internal jugular vein without thrombosis was demonstrated in one patient, Case No. 12. The father of this patient is a physician to whom the nature of the condition could be fully explained. On account of the persistence of the sepsis, and despite negative blood cultures and very slight neck swelling, consent for jugular resection was readily given. The presence of polynuclears in the vein wall and adherent lymph glands tells its own story, and throws much light on these infections.

It would seem that thrombosis of the internal jugular vein may take place in at least two ways. First, from thrombophlebitis of its small tributaries high up in the pharyngomaxillary space, especially in the region of the tonsil. Waldafel[4] is again referred to in this connection. Second, by extension of the inflammation from the sheath and from contacting lymph nodes. This was demonstrated by Case No. 12, above referred to, where there was no thrombus formation, and is interpreted as being the early pre-thrombus stage.

Secondary hæmorrhage from the small veins of the pharyngomaxillary space occurred in one patient, Case No. 11, on the second day after operation. Removal of the stiff rubber drainage tubes, and substitution of iodoform gauze promptly checked the bleeding. Subsequently soft rubber cigarette drainage material was used, and no more bleeding occurred. In this patient there also occurred paralysis of pharyngeal branches of the glossopharyngeal nerve, paralysis of laryngeal branches of the vagus, and paralysis of the spinal accessory nerve.

The following is a list of complications which occurred in these cases.

1. Rupture into the external auditory canal.....Once
2. Thrombosis of the internal jugular vein.....Twice
3. Acute mastoiditis with thrombosis of the sigmoid sinus .....Once
4. Phlebitis of the internal jugular vein without thrombosis .....Once
5. Thrombosis of the internal jugular vein and cavernous sinus .....Once
6. Thrombosis of veins of both legs followed by abscess formation .....Once
7. Thrombosis of veins in right side of pelvis followed by pelvic abscess .....Once
8. Abscess of the hip joint .....Once
9. Bilateral choked disc .....Once
10. Secondary hæmorrhage from veins in pharyngomaxillary space .....Once
11. Cystitis .....Once
12. Paresis of the ninth, tenth, and eleventh cranial nerves .....Once
13. Paresis of the twelfth cranial nerve .....Once
14. Paralysis of the pupillary fibers of the cervical sympathetic nerve .....Once
15. Blood stream infection demonstrated by positive cultures .....Twice

Extension into the chest or axilla and rupture of the carotid artery I have not yet observed.

Summary of Findings

1. Adequate drainage can be obtained only through the external route. If internal drainage has been done, and does not bring about immediate subsidence of the infection, external incision must be made without delay.

2. Phlebitis of the internal jugular vein, with or without thrombosis, is often present. There were five cases, twenty per cent. One died unrecognized; the others received adequate external drainage and lived. Evidence that involvement of the vein may occur early was shown in one of the cases that recovered. In this one there was polynuclear infiltration of the wall of the vein, erosion of the intima, and minute deposits of fibrin on the endothelium, without thrombosis.

3. Swelling of the lateral pharyngeal wall is an early and constant sign. Look for it, but do not always expect to see increased redness. It may look pale.

4. Trismus is one of the most important early signs of infection in the pharyngomaxillary space. It is due to splinting of the internal pterygoid muscle by the advancing inflammation. It is explained by the applied anatomy. The constrictor muscles of the pharynx form the inner wall of the space, and the internal pterygoid muscle a part of the outer wall. The result of infection between the two is obvious.

5. Chills, sweats, and high excursions of temperature are common symptoms, and do not necessarily require jugular resection unless they continue some days after adequate drainage has been established. They do demand immediate external drainage of the deep fascial compartments.

6. Quinsy sore throat is the diagnosis most frequently made when they come referred. When seen early the differentiation must be made, and it is sometimes difficult. It is possible for a peritonsillar abscess to antedate the infection in the pharyngomaxillary space, though I have seen none. If so, one might consider doing tonsillectomy.

7. The degree of external swelling may be small or barely distinguishable, so that by itself it is no criterion for estimating the necessity for external drainage. Absence of external swelling does not contraindicate external drainage if there is sepsis. Infection of a fascial plane may be present without visible evidence of pus. The microscope has demonstrated marked evidence of infection in just such cases. I am very much indebted to Dr. Harris P. Mosher for a slide which proves this statement.

8. Blood may remain negative despite the existence of a phlebitis, even when the blood to be cultured is withdrawn from the internal jugular vein.

9. Cervical gland abscesses usually remain superficial and localized, but occasionally the infection will spread along the layers of the deep fascia, with resulting change of picture requiring drainage of the compartments invaded.

10. Prevertebral fascia infection constitutes the well-known condition called retropharyngeal abscess. When seen early and drained before there is obstruction to breathing, recovery is

prompt. If untreated these cases die from asphyxia due to capping of the larynx by the protruding posterior pharyngeal wall, rather than from extension of the infection into the chest.

11. Torticollis from infection under the sternomastoid muscle is toward the opposite side. Torticollis from infection along the paravertebral muscles and trapezius is toward the same side.

12. The ears should be frequently inspected as there may be a coincident tympanomastoid inflammation. In this series there were two such instances, one of which went on to obstructive sigmoid sinus thrombosis.

13. The sepsis or septicopyæmia may go on to a fatal termination without apparent increase in the severity of the local manifestations. This happened in my first case, the only death in the group. The cause of death was thrombosis of the internal jugular vein and cavernous sinus. The reason, I believe, was failure to drain externally because two consultants, a general surgeon and a laryngologist, advised against it. Thrombosis of the internal jugular vein was not suspected until the signs of cavernous sinus thrombosis appeared.

14. Chills are to be expected. Blood for culture should be taken during or immediately after the first chill. Blood cultures may become negative after the vein becomes corked by the thrombus.

15. Metastatic abscesses following thromboses in distant veins are common after thrombosis of the jugular has occurred.

Conclusions

1. Cases of jugular thrombosis from infection in the planes of the cervical fascia are going unrecognized, and dying from septicæmia.

2. Absence of frank local manifestations of the presence of the infection, notably swelling, is common.

3. Delayed surgery is apt to be disastrous, and engenders regret.

I wish to acknowledge my indebtedness to Doctors P. I. McIlroy and H. vonWedel of the New Rochelle Hospital Laboratory for their skilled interpretation of the microscopic pathology in connection with the above-mentioned cases.

Cases	Age and Sex	Regional Pathology								Treatment Received	Result
		Etiology — Portal of Entrance	Pharyngo-Maxillary Space	Submaxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral — Bucco-pharyngeal — Pretracheal	Carotid Sheath	Complications		
No. 1 JENNIE R.	9 mo. f	Naso-pharyngitis — Infected adenoid				Retro-pharyngeal abscess			None	Per-oral incision	Recovery
No. 2 ROSE A.	8 mo. f	Infected tonsils and adenoid				Retro-pharyngeal abscess			None	Per-oral incision	Recovery
No. 3 JOSEPH H.	6 yrs. m	Infected tonsils and adenoid				Retro-pharyngeal abscess			None	Per-oral incision	Recovery
No. 4 MARY F.	42 yrs. f	Hypopharynx trauma					Bucco-pharyngeal abscess		None	Incision through laryngoscope	Recovery
No. 5 OSCAR B.	19 mo. m	Infected tonsil	Lateral pharyngeal abscess						None	Per-oral incision	Recovery
No. 6 KATH. R.	20 yrs. f	Pharyngitis and infected tonsils	Pharyngo-maxillary cellulitis						None	Non-surgical treatment	Recovery
No. 7 CENTER H.	14 mo. m	Infected tonsils and adenoid	Pharyngo-maxillary abscess	Submaxillary and Parotid	Secondary				Rupture into ext. auditory canal	External drainage	Recovery
No. 8 LEONARD P.	16 yrs. m	Infected tonsil remnant	Pharyngo-maxillary abscess		Secondary			Sheath infection	Thrombosis of internal jugular vein and cavernous sinus	Per-oral incision	Death
No. 9 GILBERT K.	3 yrs. m	Infected tonsils	Pharyngo-maxillary abscess		Secondary with abscess				Lymph glands abscess	External drainage	Recovery
No. 10 JANE M.	5 yrs. f	Pharyngitis and infected tonsils	Pharyngo-maxillary abscess secondary	Submaxillary and parotid infection secondary	Cervical glands abscess primary				None	External drainage	Recovery

No. 11 SHAW C.	17 yrs. m	Pharyngitis	Pharyngo-maxillary abscess						Secondary hæmorrhage — Paresis of 9th, 10th and 11th cranial N's	External drainage	Recovery
No. 12 SALLY H.	4 yrs. f	Trauma from adenoid operation	Pharyngo-maxillary abscess		Glands on sheath infected			Infected	Phlebetis of internal jugular	Ext. drainage and resection of jugular vein	Recovery
No. 13 DOR'THY S.	19 yrs. f	Otitis media and petrous temporal bone infection	Pharyngo-maxillary abscess	Parotid fascia infection					None	External drainage	Recovery
No. 14 ROSE F.	32 yrs. f	Infection in zygomatic fossa following local anæsthesia injection	Pharyngo-maxillary abscess	Parotid and submax. fascia infection					None	External drainage	Recovery
No. 15 EDYTHE R.	26 yrs. f	Submaxillary lymph node suppuration	Pharyngo-maxillary abscess — secondary	Submaxillary space abscess — primary				Carotid sheath infection	None	External drainage	Recovery
No. 16 ALFRED S.	7 yrs. m	Rhino-pharyngitis	Pharyngo-maxillary abscess	Submaxillary space infection	Lymphadenitis secondary				Mastoiditis — Sigmoid sinus thrombosis — Hip joint abscess — Paresis of 12th nerve — Paresis of pupillary fibers of cervical sympathetic nerve	Ext. neck drainage — Mastoidectomy — Drainage of sigmoid sinus — Resection of int. jugular vein — Hip joint drainage	Recovery
No. 17 ELIZ. W.	6 yrs. f	Pharyngitis			Cervical gland abscess				None	External drainage	Recovery
No. 18 JOSEPH C.	10 mo. m	Infected tonsils and adenoid			Cervical gland abscess				None	External drainage	Recovery
No. 19 SYLVIA F.	14 yrs. f	Trauma from tonsil operation	Small localized abscess beneath tonsil fossa muscle bed						None	Spontaneous drainage	Recovery

Cases	Age and Sex	Regional Pathology								Treatment Received	Result
		Etiology — Portal of Entrance	Pharyngo-Maxillary Space	Submaxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral — Bucco-pharyngeal — Pretracheal	Carotid Sheath	Complications		
No. 20 EDGAR W.	21 yrs. m	Dental infection		Submaxillary fossa infection — Parotid fascia infection					None	External drainage	Recovery
No. 21 CHARITY D	53 yrs. f	Thyroid gland fascia-Lymph node			Lymphadenitis on visceral layer		Pretracheal or visceral fascia abscess		None	External drainage	Recovery
No. 22 DANIEL M.	30 yrs. m	Dental infection		Submaxillary and submental triangles Infection with infection of floor of mouth					None	External drainage	Recovery
No. 23 ROSE L.	7 yrs. f	Dental — — Alveolar abscess		Submaxillary and submental triangles — Infection floor of mouth					None	External drainage	Recovery
No. 24 LOUISE B.	47 yrs. f	Pharyngitis		Submaxillary and parotid fascia, and carotid sheath	Secondary — Without suppuration			Carotid sheath infection	Thrombosis of internal jugular veins — Thrombosis of veins of both legs with abscess formation — Thrombosis of pelvic veins followed by pelvic abscess — Bilateral choked disc	External drainage of Carotid sheath — Leg abscess drainage bilateral — Pelvic abscess drainage	Recovery

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DISCUSSION

Dr. T. E. Carmody, Denver: I think this is a very timely paper, and the review of the literature is also timely.

Doctor Beck has called our attention to the fact that most of these infections are from the region of the tonsils. My experience differs in that most of the cases I have seen have been from the region of the teeth, although one came from the ear and two from the thyroid gland.

He speaks also of jugular thrombosis, which seemingly is a late manifestation in too many cases simply because of neglect. I believe a good many cases go unrecognized.

As to the kind of infection, he speaks of the streptococcus hemolyticus predominating. We have found the staphylococcus, the streptococcus, and mixed infection very frequently.

The treatment obviously is to drain early. I observed a case some years ago in which a general practitioner was applying poultices. I suggested that if he kept on poulticing it would open about three days after death. Continued poulticing predetermines in a great many instances a state where they die a few hours after operation has been done. It does not make any difference whether the operation is thorough or not, the patient is too septic to respond.

The sign which Dr. Beck called attention to, the failure to open the mouth, very frequently indicates that the infection is high up around the internal pterygoid muscle. He also mentioned infection along the stylomandibular and the stylohyoid ligaments. Treatment by internal drainage seldom will be of any value except where there is perforation of the pterygo-maxillary fossa.

I would like to show a few slides, some of them supplementing those of Dr. Beck, and some the cases I have seen.

Dr. Harris P. Mosher, Boston: This paper is one of the most important which has appeared in the field of laryngology for many years. It is based on a large number of cases. That there should be only one

death out of twenty-four patients is a surgical triumph. The cases are grouped and analyzed in a masterly way.

The writer's conclusions should be burned into the memory of every laryngologist and every general surgeon. Unfortunately, the general surgeon seems to know less about this condition even than the laryngologist.

The conclusions of the paper are: Cases of jugular thrombosis from infection within the planes of the cervical fasciæ are often unrecognized. I should supplement this by saying that they are unrecognized daily. Local or external swelling is often absent. Delayed surgery is disastrous. Sepsis, especially continued sepsis, is the great guiding point.

The writer's exposition of the anatomy is splendidly clear. I envy him his diagrams.

I will mention a few individual points in his paper and then speak of his grouping of cases. He starts off with the statement that most cases in the beginning pass for quinsy sore throat. I heartily agree to this. Absence of a positive blood culture, he states, is probably due to a blocking off of the vein, and he stresses the point that the surgeon should not wait for a positive blood culture. Trismus, due to splinting of the internal pterygoid muscle when pus is present high in the neck, I have never seen emphasized before. In regard to swelling of the floor of the mouth of unknown origin, I should advise thinking of abscess at the base of the tongue. It is odd that there is no case in his series of Bezold's abscess. It is not uncommon for such an abscess to drain into the pharyngo-maxillary fossa. Anatomically, of course, abscess of the petrous tip also can drain into the pharyngo maxillary fossa, and Dr. Beck has had one such case. I have seen one. The writer advises a generous incision in seeking deep pus in the neck. I have seen operators fail because of the lack of it. Timid incisions breed timid surgery.

The high spot of the paper to me is his grouping and analysis of the sites of infection. This is the first paper to dwell on the regional grouping of infections of the neck. It is a most valuable addition to the study of the subject. I urge that every laryngologist make himself thoroughly familiar with it. In his analysis and grouping, he points out which type of case is most liable to infect the internal jugular vein, and which type does this relatively rarely. For instance, he points out that every swelling of the side of the neck does not call for exposure of the internal jugular vein, although of course, any swelling may come to this. Cases of infection of the submaxillary space are relatively simple, as we all know. In such cases it usually comes from the teeth. Ordinary or straight retropharyngeal abscess is readily dealt with in most cases by an incision carried out within the pharynx. Further, the writer states that suppurating glands of the neck usually do not lead to thrombosis of the internal jugular vein. Again, of course, I agree.

The main thing the writer stresses in the paper is that the laryngologist should be on the watch for these cases and have the surgical courage to treat them properly. The degree of sepsis is the one great guide. The longer I deal with deep cervical abscess of the neck or watch my confreres deal with it, the more convinced I am that without a knowledge of

the fundamental anatomy of the surgical compartments of the neck — and it is really very simple — and without a knowledge of the possibilities of the submaxillary approach to deep pus in the neck, a laryngologist cannot be classed as a safe practitioner of the specialty.

Dr. John F. Barnhill, Miami Beach, Fla.: The paper needs very little discussion. It needs only emphasis.

It is unfortunate that our special societies have not long ago given greater attention to this type of surgery, which lies in our field. Too many laryngologists on finding a deep abscess in the neck turn it over to a general surgeon, or avoid attacking it surgically too long, and so I wish to add to the emphasis already given that delay is dangerous.

In looking up the old literature on what surgeons did one hundred and fifty years ago, I find death was very common in pharyngeal and cervical abscess. The first President of the United States died of this infection, with old Dr. Craik standing by helpless to avert the end. So great has been the progress in surgery of the neck that there are probably one hundred surgeons in this room who would have saved General Washington's life on that occasion.

The essayist said one thing to which I wish to take some exception. He said we should wait for fluctuation of the neck before incision. I may have misunderstood him, but that was my understanding. I wish to submit to you that fluctuation is one of the late symptoms, so late that we should not wait for it. The reason it is late is that the abscess is covered by dense fascia and by overlying muscles; it is in a tough bag and cannot readily escape. One does not readily find it. The last speaker said something about that. There is not always notable swelling and hence one must often determine whether there is an abscess by other symptoms, and one who cannot do that is weak in his diagnostic powers.

It has been emphasized that the opening into the cervical abscesses should be widely made. There are good reasons for that, one of which is that the operator encounters numerous veins, and if he gets a good deal of bleeding, as he is apt to do from open veins, he may not be as thorough or safe as he would wish. A wide incision is necessary to avoid veins, and to distinguish important structures as our course comes into the deeper parts. Then if one must deal with the jugular vein a wide operative field is almost essential.

I would also emphasize a few of the ailments for which the infection under discussion may be mistaken. It may be mistaken for a ranula that has gone for a good while and has broken through the floor of the mouth into the submaxillary space. I have recently seen such a case which was so mistaken. There are other affections which give rise to difficulty of differentiation. A midline tumor, a thyroglossal cyst, has been so mistaken. One is not likely to make that mistake, but I have seen it made. I therefor emphasize the fact that we have other affections of the neck which, if the history of the case is not carefully taken into account, may lead to error in diagnosis. If one explores the pharyngo-maxillary fossa with the finger, as he should do, unless the fossa has already ruptured

outwardly, the finger may not always enter the abscess cavity, the operator mistaking the outer fascial wall of the abscess cavity, not penetrating farther and therefore missing the abscess. The fascia in this region is very dense and tough, and unless it is broken down by necrosing action of pus, one may have trouble in finding it.

Otologists should do this type of surgery. Dr. Mosher has emphasized the necessity of learning the anatomy, and that is important. But he says it is simple. It is simple for Dr. Mosher. To me it is not simple, but any who are willing to work hard enough on the cadaver may learn it.

Dr. T. C. Galloway, Evanston, Ill.: The presentation has been very thoroughgoing on an anatomical basis both by the essayist and the discussers. Something more, however, might be said about infective organisms. Davis and Pilot have found the organism of Vincent-Plaut in 85 per cent of tonsils and in many other infections around the head and neck. In a series of nine severe deep neck infections we found in pus from the depth, spirilla and fusiform bacilli in six. In searching for these organisms it is important to remember that they cannot be demonstrated by ordinary culture; the culture must be a proper anerobic one or the organism demonstrated by smear.

In such infections around the head and neck especially from the teeth we have been administering arsenicals early. Formerly we saw five to eight of the severe type a year at the Cook County Hospital but since this routine we have seen only one or two such cases annually. I cannot but feel that the organisms of Vincent are very important and that if arsenicals are properly given the extensive anatomical dissections may often be avoided.

Among other factors to remember is that these patients are often dehydrated on account of difficulty in swallowing and the fluid intake should be checked. Intravenous glucose may be life saving here.

Another thing to remember is that when sudden and profuse hemorrhage occurs in a retro- or para-pharyngeal abscess almost certainly the internal carotid artery has been eroded. I had such a case which might have been saved had I realized quickly enough the need of immediately ligating the internal or common carotid.

Dr. August L. Beck, closing: Dr. Barnhill brought up the question of fluctuation. The complete paper goes into considerable detail on that point, more than I could speak of from the platform because of lack of time. I did, however, differentiate between the superficial gland infection and deep gland infection. In infection of the superficial glands such as occurs in children, there usually is not any sepsis and one is justified in waiting for localization in cases of that type. In the deeper gland involvements one must learn to determine just when the sepsis is getting to the danger point and drain early, not waiting for fluctuation.

Dr. Carmody mentioned that the bacteriology in his cases showed a predominence of staphylococcus. This may be due to the fact that his cases were largely of dental etiology. Only three of my cases were of dental origin.

Dr. Galloway also spoke of the bacteriology. That is covered somewhat fully in the paper and is tabulated. I just drew the most important conclusions from our findings. Smears were taken if seen before the neck complication was already existent. I always have in mind the possibility of Vincent's Angina infection, and always apply the arsenical preparations locally in every postoperative tonsil case, except in small children and infants. I have not encountered any Vincent's in these neck infections. It must be recalled that I get most of these cases when they are full-blown, and at that time they probably have mixed infection of the cervical glands.

We must guard against dehydration, and employ all therapeutic aids including transfusion. The possibility of arterial rupture is mentioned in the complete paper. I have not had such a case. I have had a good many interesting complications which I said nothing about in the paper as read.

I am very grateful to the gentlemen for their discussion. They have really made the paper.

As the paper developed and the facts became correlated, I felt at times much as a myope must feel when he wears his first pair of correcting glasses.

SYMPOSIUM

NASAL MUCOUS MEMBRANE

I. THE HISTOLOGY AND CHRONIC INFLAMMATION OF THE NASAL MUCOUS MEMBRANE

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HISTOLOGY

The essential tissues comprising a mucous membrane are (1) a surface epithelium, (2) a basement membrane and (3) a tunica propia or stroma. In addition, there may be, (4) a muscle

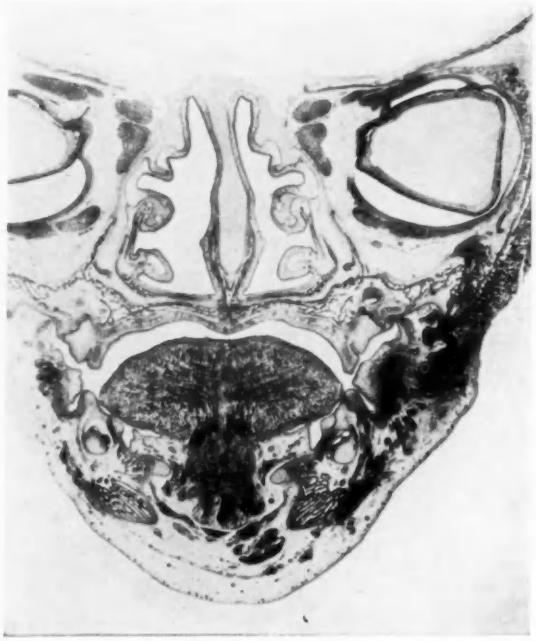

FIG. 1—Nasal fossæ of 4 months fœtus. The epithelium is of the pseudo-stratified ciliated variety.

coat, the muscularis mucosa, and (5) a submucosa. Of the mucosa membranes of special interest to the rhinolaryngologist, only the esophagus and lacrymal duct possess these additional coats.

The epithelium of the mucous membrane may be of any of the varieties of epithelial cells. According to the shape of the cell it may be classified as squamous, cuboidal, columnar or transitional. According to the arrangement of the cell it may be stratified or pseudo-stratified.

The basement membrane is a band of unstained or faintly staining tissue, collagenous in character, on which the epithelial

FIG. 2—Nasal fossæ of an 8 month child. The epithelium is still of the ciliated variety.

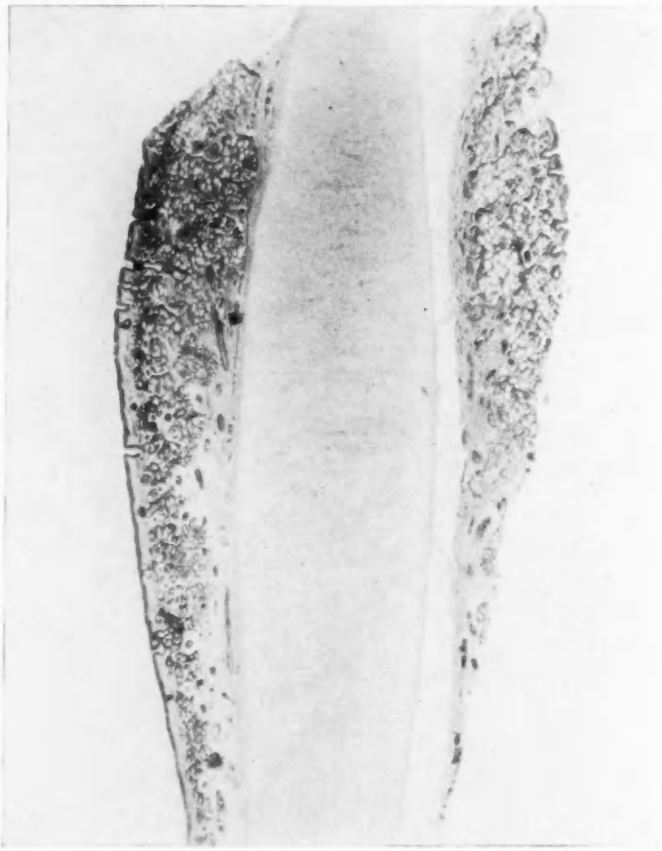

FIG. 3—Nasal septum — adult. Characterized by many glands in the stroma, particularly in the region of the tubercle.

cells apparently rest. Its origin is uncertain. From the fact that it takes the connective tissue stain it is thought to be derived from the underlying connective tissue. It varies in thickness; it may be so delicate as to appear absent; in chronic infections it may be tremendously thickened. It is cribiform. These canniculi permit processes of the underlying connective tissue cells to extend into the epithelium and they permit passage of cells to the surface.

The tunica propria or stroma consists of a fibro-elastic connective tissue network, which serves to attach the membrane to

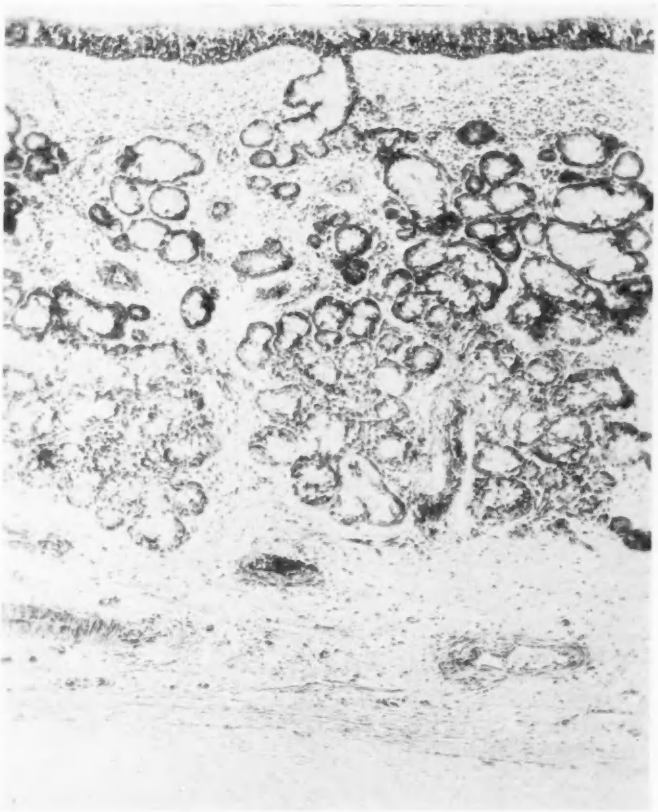

FIG. 4—Nasal septum—region of tubercle, showing the profusion of glands.

the underlying structures. Superficially the stroma is loose, rich in cells, but as it deepens it becomes more compact and condensed, with the progressive appearance of elastic fibers. Over bone or cartilage this condensation of the stroma forms, respectively, the periosteum or perichondrium.

Of the cellular elements; lymphocytes, polymophonuclear leukocytes, plasma cells, eosinophiles, connective tissue cells, elastic fibres; the lymphocyte predominates. These may be scattered throughout the tissue as diffuse lymphoid tissue; or may be gathered into a compact mass to form a distinct lymph-node or nodule.

The stroma supports the glands. These vary, from the simple straight tubule lined with goblet cells, to the tubo-alveolar type of gland.

The blood supply is by way of vessels which enter deep in the stroma. These vessels send branches through the propria to form a dense capillary network beneath the epithelium, and around the neighboring glands. The venous return is by way of superficial blood spaces which lead to a deeper venous plexus. In certain areas of the respiratory mucosa these plexuses are so greatly developed as to form cavernous sinuses and assume the character of erectile tissue.

Such then is the basic histologic picture of any mucous membrane. The nasal mucosa shows characteristic variations according to site.

The Nasal Septum — In the embryo and infant, the epithelium is of the pseudo-stratified ciliated variety. In the adult, due to the traumatization incident to life, the exposed portions of the septum, frequently show nature's protective measure, in that the pseudo-stratified ciliated epithelium is replaced by the stratified squamous variety. The basement membrane is distinct. The stroma is characterized by the tremendous number of mucous and serous glands. In the region of the tubercle of the septum there appear large blood lakes or cavernous spaces. Lymphocytic infiltration of the stroma is marked. In places the lymphoid cells form distinct lymph nodules.

Over the cartilage and bone, the stroma becomes condensed to form the perichondrium or periosteum. At the junction of the cartilage and bone, fibers of the stroma interlace with fibers from the opposite side.

The epithelium of the olfactory portion is of the stratified variety, the surface cells being both sustentacular or supporting cell and the olfactory cell.

The sustentacular cells present three distinct parts.

(1) A superficial portion, which is broad and cylindrical containing pigment and granules arranged in longitudinal rows. These cells have well-marked straight thickened free borders, which unite to form the so-called "membrana limitans olfactoria."

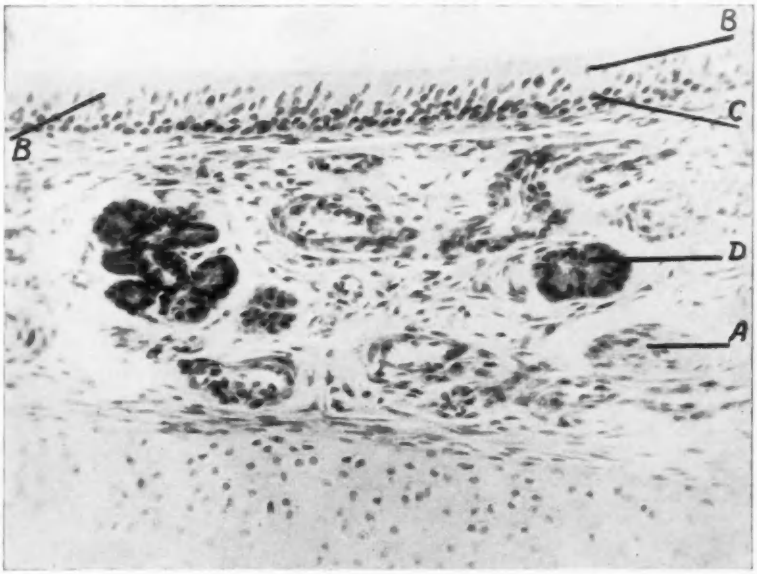

FIG. 5—Olfactory mucous membrane of cat: A, Nerve bundle; B, Oval nuclei; C, Round nuclei; D, Glands of Bowman.

(2) A middle portion which contains an oval nucleus. As the nuclei of these cells all lie on the same plane, they form a distinct narrow band — "the zone of oval nuclei."

(3) A thin filamentous process which extends from the nuclear portion down between the cells of the deeper layers.

The olfactory cells lie between the supporting cells. Its nucleus is spherical, they lie at different levels and are more deeply placed than those of the sustentacular cells; they form the so-called "zone of round nuclei." The olfactory cell sends a dendrite which extends to the surface and an axone, which collects with other axones to form eighteen to twenty-four bundles to pass through the cribiform plate to the olfactory bulb.

Extending from the surface cells, to the basement membrane are the basal cells. They are small, nucleated cells, with irregular or branching protoplasm which anastomoses with that of neighboring basal and sustentacular cells.

The basement membrane of the olfactory portion is not well-developed.

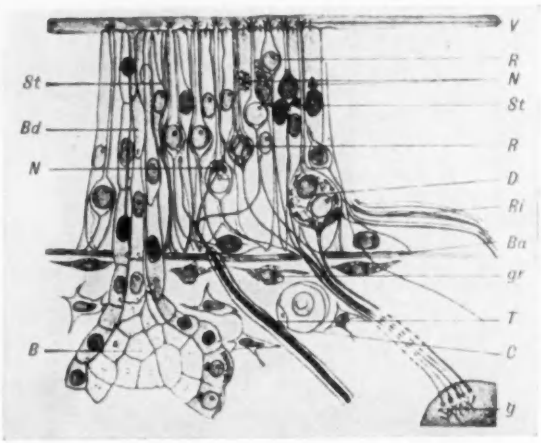

FIG. 6—Diagram of the olfactory mucous membrane of man: *St*, Sustentacular cells with tono-fibrils; *R*, olfactory cells with neurofibrils and the olfactory vesicles, *V*; *D*, large binucleated olfactory cell; *Ri*, olfactory fibers; *T*, myelinated fiber of the trigeminus; *g*, olfactory glomerulus; *B*, gland of Bowman, with diplosomes in its cells, and with its excretory duct, *Bd*; *gr*, granular connective tissue cells; *Ba*, layer of basal cells; *N*, Golgi net in the sustentacular and in the olfactory cells; *C*, capillary. After Kolmer.

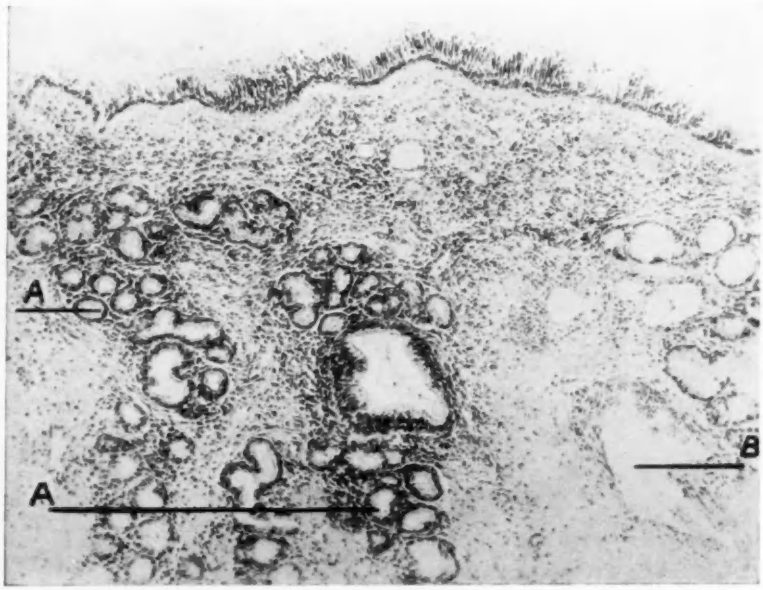

FIG. 7—Middle turbinate—100×. A, Glands; B, Cavernous sinus.

The tunica propria is delicate, loosely arranged. Embedded in the stroma are a large number of simple branched glands — the "glands of Bowman." The secretory cells of these glands are large and irregular and contain a yellowish pigment. These glands, described as serous, are now believed to be mucous.

The Middle Turbinate — The mucosa of the middle turbinate varies greatly in thickness. The ciliated epithelium is frequently replaced by a low cuboidal or squamous variety over the anterior tip. The stroma is particularly rich in glands, especially over the posterior tip of the turbinate. The periosteum is firmly attached to the underlying bone.

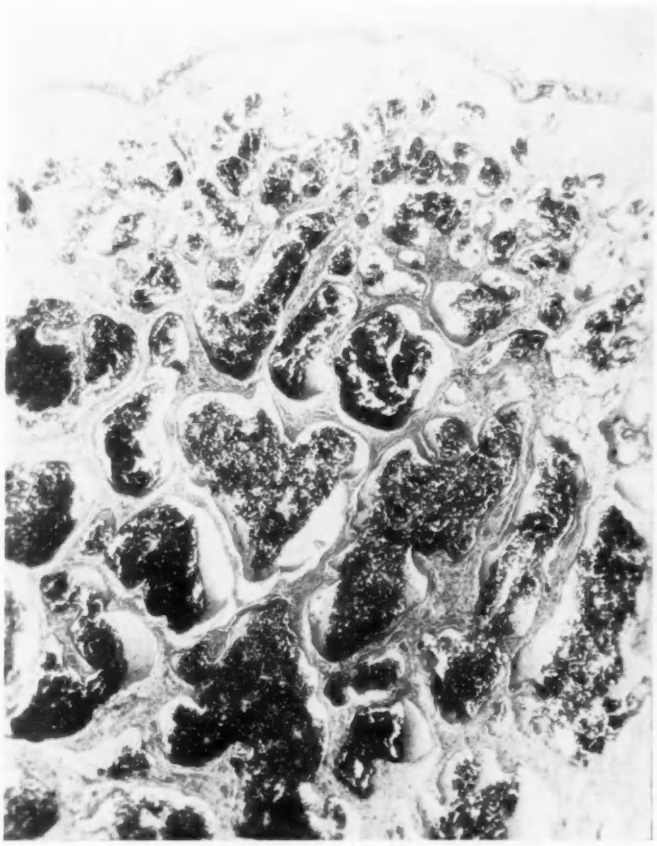

FIG. 9—Inferior turbinate with dilated swell bodies. 30×.

Inferior Turbinate — Like the middle turbinate the epithelium of the anterior tip and inferior border of the inferior turbinate in the adult, may be replaced by a low cuboidal or squamous epithelium. The basement membrane is usually distinct. The stroma is characterized by the pronounced number of blood lakes or cavernous sinuses. Glands are likewise numerous, but not to the extent of the middle turbinate.

Maxillary Antrum — The mucosa of the normal maxillary sinus is a thin delicate structure. The epithelium is continuous with that of the nose — of a pseudo-stratified ciliated variety, containing numerous goblet cells. The basement membrane is fairly distinct. The stroma is poorly cellular. The glands are scattered, but are most numerous in the region of the osteum. The periosteum strips readily from the underlying bone.

Ethmoid — The mucous membrane of the ethmoid cells resembles that of the maxillary sinus. The epithelium is likewise ciliated columnar variety with an occasional goblet cell. The stroma is thin and delicate, containing but a few glands. It is more firmly attached to the underlying bone than that of the maxillary antrum.

Frontal-Sphenoid — The mucosa of the frontal and sphenoid is very similar to that of the ethmoid. Stratified, ciliated epithelium, faint basement membrane, stroma delicate, few glands, but fairly firmly attached to the underlying bone.

Pathology

Such then, is the histologic picture of the normal nasal mucous membranes. Let us review the changes that occur when this normal is altered by inflammation. First, let us limit and simplify our definition of inflammation as "the tissue reaction to an irritant." The changes that result are (1) those of the blood stream and (2) those of the cellular elements.

Most pathologists are agreed that the alteration in the blood stream are first a temporary constriction of the vessel with an accelleration of the blood stream. This is followed by a dilation of the vessel and subsequent slowing of the current of the blood.

Examination of the blood, as it flows along through the vessels, shows that there is normal central, axial zone, containing

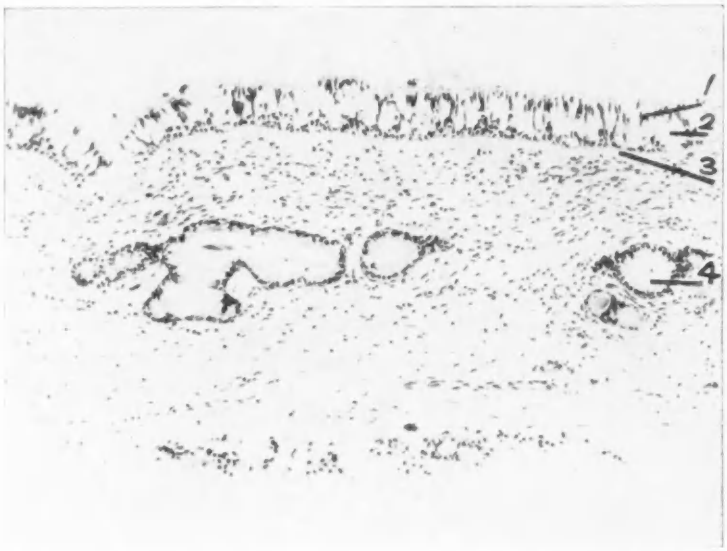

FIG. 9—Normal maxillary sinus mucos—100×. 1, Pseudo-stratified, ciliated epithelium. 2, Goblet cells. 3, Basement membrane. 4, Glands.

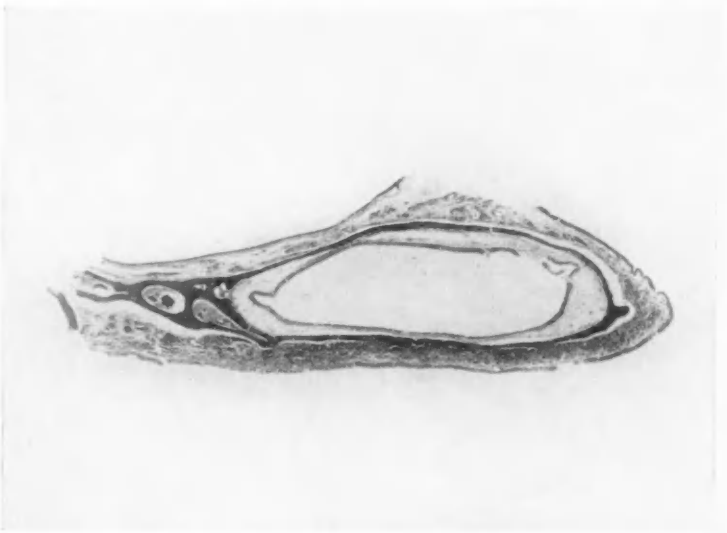

FIG. 10—Ethmoid cell—10×.

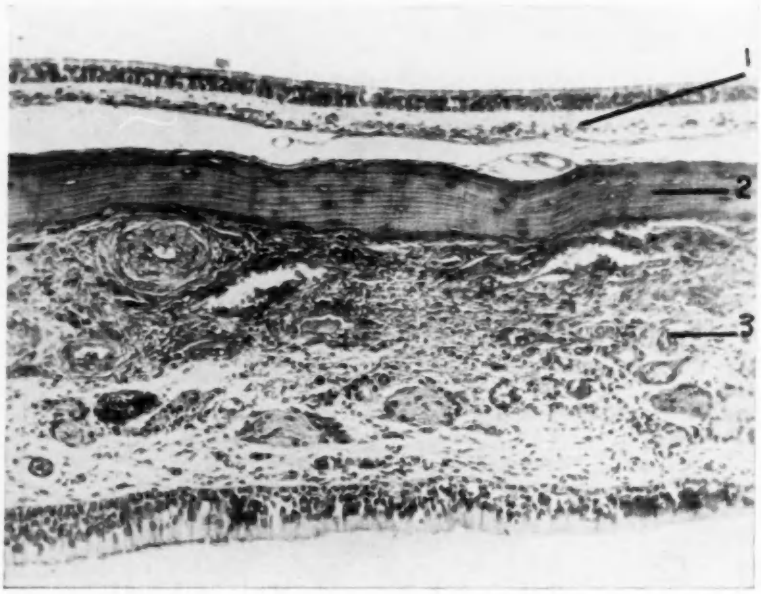

FIG. 11—Ethmoid—140×. 1, Mucosa of ethmoid. 2, Bone. 3, Nasal mucosa.

the blood corpuscles; and, in addition, there is, between the axial zone and the capillary wall, a peripheral zone containing blood plasma. These zones are maintained by the vortical force acting upon the heavy cells as compared with the lighter plasma. In normal circulation or in the early stages of inflammation when the vessel wall is constricted, these zones are clearly maintained. With the dilatation of the vessel wall and slowing of the circulation, the lighter cells of the blood stream, due to the lowered specific gravity, appear in the peripheral zone. During this stage the leukocytes can be seen lagging along the vessel wall adhering, and finally by diapedesis pass through the wall into the surrounding tissue. There they migrate toward the point of injury. At the same time the fluid of the blood stream passes through the vessel wall and appears in the tissues. This then is the first reaction of inflammation. The fixed tissues react to the inflammation by a migration of the fixed tissue cells, particularly the connective tissue cells.

In mucous membranes, as there is a dense capillary network just beneath the basement membrane, it appears reasonable to

expect that the most marked change occurs primarily in this region. In acute inflammations of mucous membrane there is, therefore, a dilatation of the tissue or lymph spaces just beneath the epithelium, with an increase of the cellular elements. As the infection passes from the acute to the chronic stage, the polynuclears disappear so that of the cellular elements the small lymphocyte predominates.

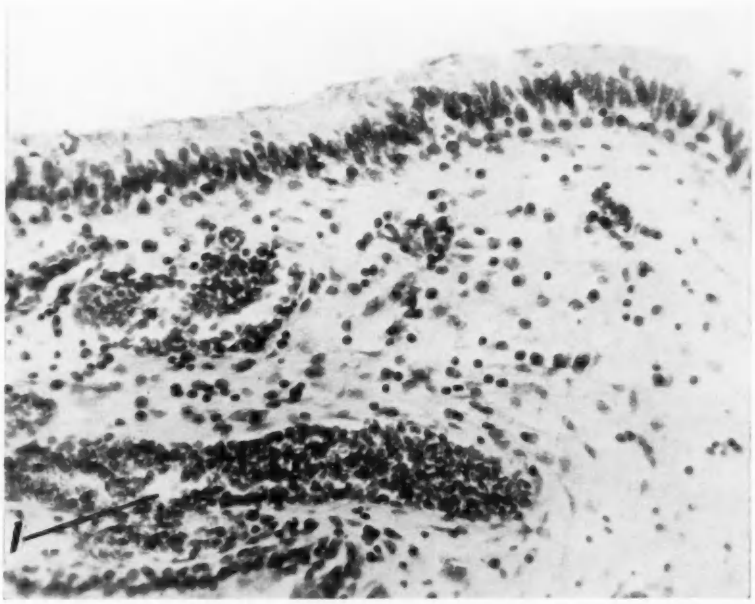

FIG. 12—Antral mucosa—300×. Acute inflammation. 1, Dilated capillary.

The tissue reaction of chronic inflammation is, therefore, a thickening of the blood vessel wall, with an increase of the cellular elements of the tissue. These cellular elements are: the lymphocytes, plasma cells, connective tissue cells and endothelial cells. In some conditions, such as asthma, hay fever and the parasitic infections, the eosinophile may show a marked increase in number.

I agree most heartily with Mullin and Ball[1] in that the pathologic findings of chronic sinusitis gives no clue as to the clinical history of the case. According to the microscopic pic-

FIG. 13—Edematous mucous membrane.

FIG. 14—Chronic sinusitis—60×. Infiltrative type—showing marked cellular reaction.

ture, I have classified chronic inflammation of the sinus mucous membrane,

1. Edematous
2. Infiltrative
3. Fibrotic
4. Cystic
5. Degenerative

Edematous — The predominate microscopic picture is, as the name indicates, that of edema. This edema is most marked, as would be expected from the dense capillary network beneath the epithelium, in the superficial portion of the stroma. The cellular elements are not prominent. The vessel walls are thickened and the glands are dilated.

FIG. 15—Chronic sinusitis—45×. Polypoid infiltrative type.

Infiltrative — In the infiltrative type of chronic inflammation there is a cellular predominance consisting principally of the lymphocytes. This infiltration is throughout the stroma particularly beneath the basement membrane but is most marked around the glands. In certain areas the lymphocytic infiltration is so pronounced as to form or resemble lymph-nodules. Most text books speak of this lympho-cytic infiltration as being most marked around the blood vessels. It is true that there is a perivascular infiltration but I have been very much impressed by the marked periglandular infiltration.

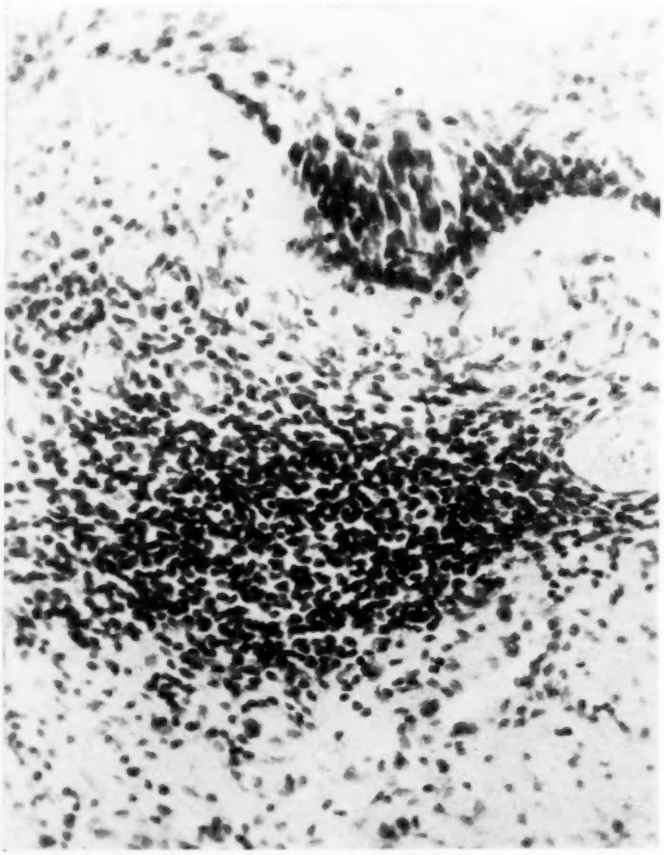

FIG. 16—Chronic infiltrative sinusitis—300×. Predominating cell being the lymphocyte; with an increase of plasma and connective tissue cells.

FIG. 17—Chronic sinusitis—asthmatic—300×. Great increase of the eosinophiles.

In the chronic inflammations the glands are exceedingly numerous. Whether this is an actual increase in glands or a great activity of glands which are normally present so that they are dilated and easily localized, I am unprepared to say. The blood vessels show thickening.

Fibrotic — In the fibrotic type of chronic inflammation the fibrosis predominates. There is a marked increase of fibrous tissue with constriction of the glands and a marked decrease of the cellular elements. I have been unable to find many specimens showing minute abscesses deep in the stroma as reported by some authors.

Cystic — The subject of cysts of the antral mucous membrane has been covered most thoroughly by McGregor.[2] Whether these cystic changes are due to a blocking of the ducts of the glands by occlusion of the lumen or by a choking of the lumen by the periglandular infiltration is open to debate. Perhaps, there is an element of truth in both theories. Under the microscope, the picture is that of many cysts.

FIG. 18—Chronic sinusitis—30X. Fibrosis.

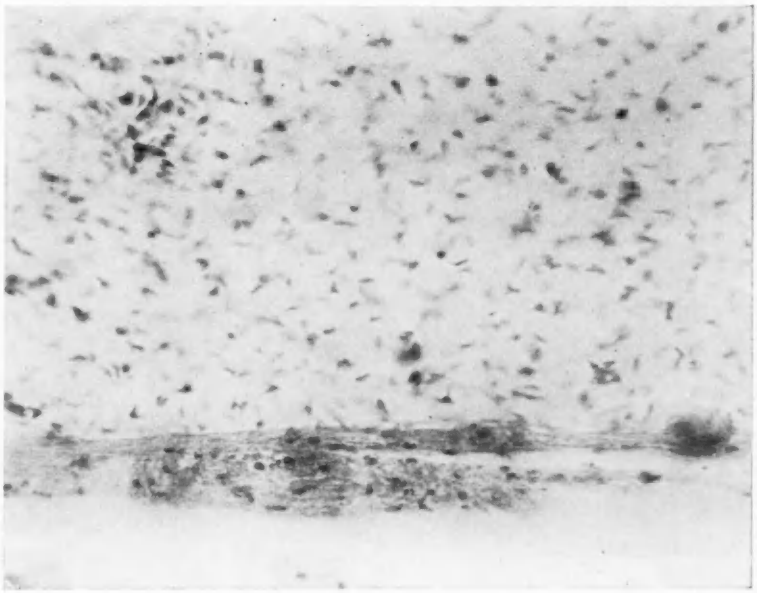

FIG. 19—Chronic sinusitis—300×. Fibrosis.

Degenerative — The term — degeneration of the antral mucous membrane — has crept into our rhinological literature, but the term is vague. True, degenerative changes of the antral mucous membrane in my experience, are rare. I have found but three such specimens. The term should be enforced by a descriptive adverb.

I am not convinced that the epithelial cells in chronic inflammation are easily destroyed. I do not believe that the loss of cilia with desquamation of the epithelial cells plays a prominent part in the chronic inflammations. I have been amazed to find structurally perfect ciliated epithelial cells in pus antra of long standing. My conviction has been confirmed by Doctors Mosher and Ross in their vital stain studies.

Turbinates — The turbinate mucous membrane change is that of mucous membranes elsewhere in the nasal cavity. The same pathologic process of edema, fibrosis, infiltration and cystic formation holds true.

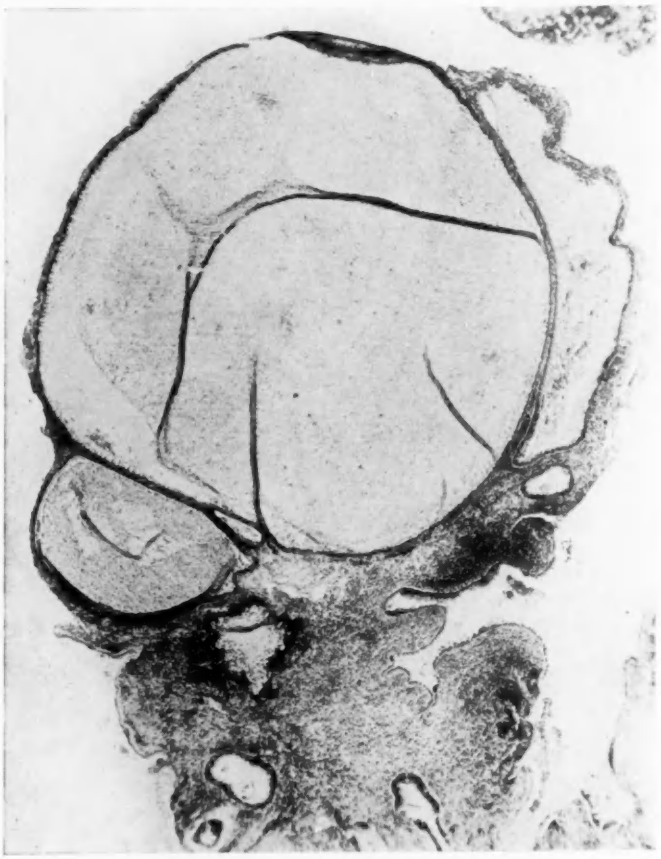

FIG. 20—Chronic sinusitis—25×. Cystic.

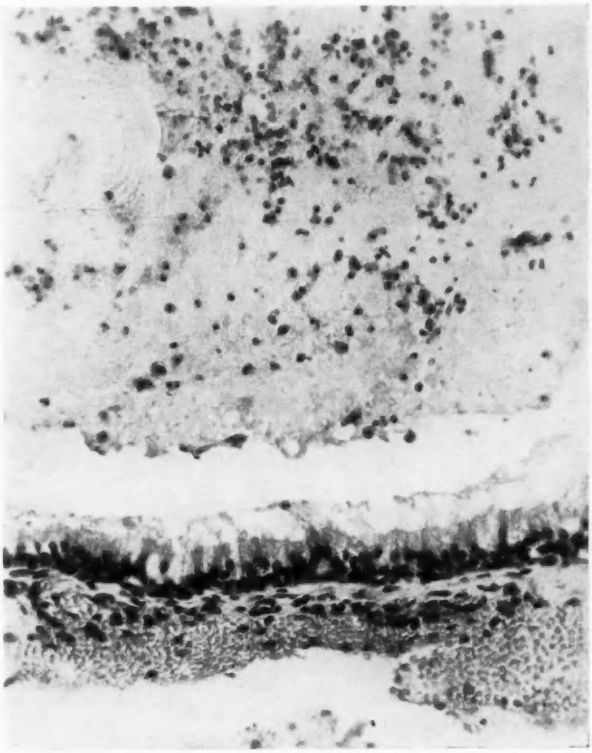

FIG. 21—Chronic sinusitis—250×. Showing infected cyst.

The terms hypertrophy and hyperplasia as applied to the turbinates frequently create confusion as to the pathologic process.

Hypertrophy, meaning in its truest sense, an increase in size, but not in numbers of the composite parts, may be physiologic or pathologic.

An irregular papillary hypertrophy frequently occurs along the inferior margin of the inferior turbinate or over the posterior tip of the middle turbinate.

Hyperplasia designates an increase in number of one or more tissue elements. In the turbinate this hyperplasia is usually of the fibrous tissue — so that the picture is that of fibrosis.

FIG. 22—Fibrotic turbinate with cyst formation.

Nasal Polyps — It is generally conceded that the etiologic factor in nasal polyps is inflammation. At one time they were considered as being myxomatous in character, but are now considered as being of an overgrowth of tissue normal to their region of origin. In other words, they show all the elements of the mucous membrane from which they spring and their changes are those of mucous membrane. The varieties of polyps are then: (1) edematous, (2) fibrous and (3) cystic or a combination of these types.

The epithelium covering a polyp is that normal to the region in which it arose. As the polyp grows and incident to traumatization of pressure, the epithelium may become thinned to a squamous-like layer of cells or may show true stratified squamous characteristics.

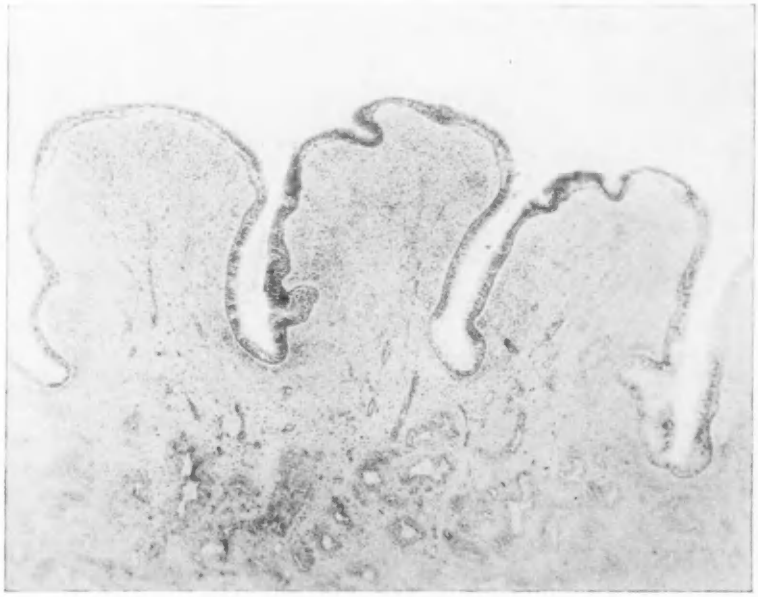

FIG. 23—Papillary hypertrophy of inferior turbinate.

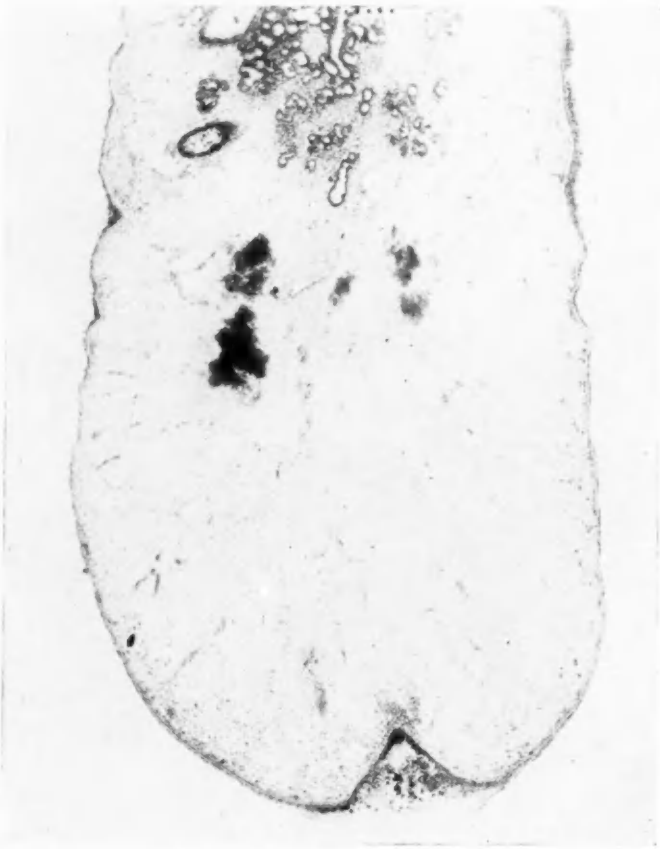

FIG. 24—Nasal polyp—30×. Showing all the elements of a mucous membrane—epithelium, basement membrane and stroma containing blood vessels and glands.

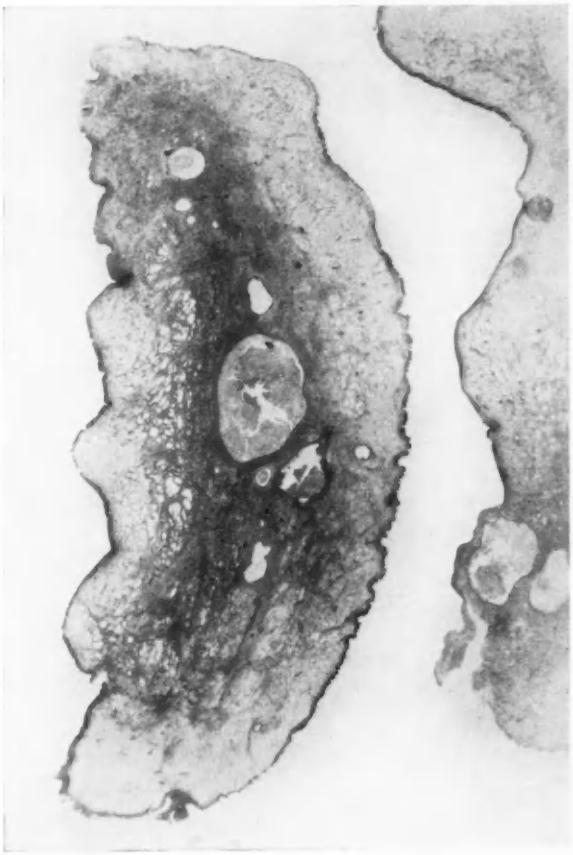

FIG. 25—Nasal polyp—cyst formation.

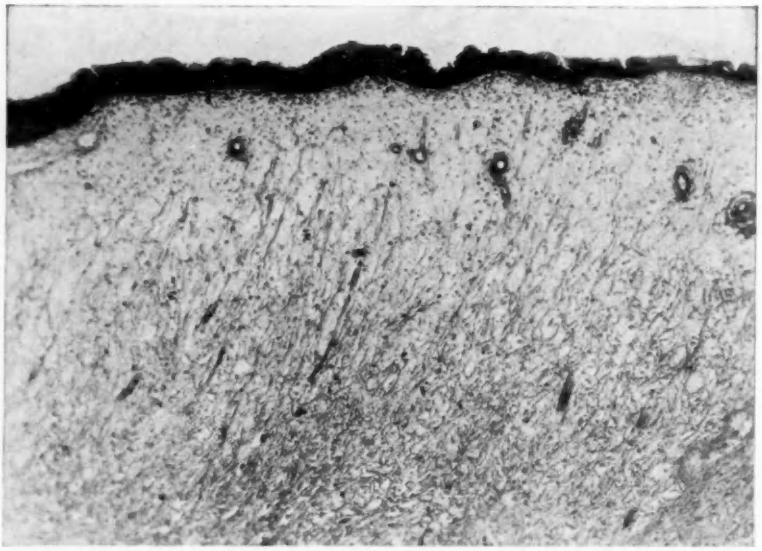

FIG. 26—Nasal polyp—80X. Transition of epithelium.

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2. McGregor, G. W.: "The Formation and Histologic Structure of Cysts of the Maxillary Sinus." Archives of Otolaryngology. Vol. 8, No. 5. November, 1928, p. 505.

NOTE — I wish to express my appreciation to Dr. Werner Mueller for the microphotographs illustrating this paper and to Dr. P. E. Ireland for the colored slides.

II. THE REFORMATION OF MUCOUS MEMBRANE IN TWENTY REOPERATIVE CASES OF CHRONIC MAXILLARY SINUSITIS

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THE WHOLE surface of the body and all the body cavities that are exposed to the air are covered with epithelium. The keeping of this surface intact is one of nature's greatest responsibilities, and she will accomplish this not only without our help, but in spite of us. The antrum is no exception.

The following twenty cases prove that reformation of mucous membrane does take place in the antrum after the radical operation for chronic maxillary sinusitis. That it regenerates as normal mucous membrane over the whole of the antrum no one can say without examining every millimeter of the mucous membrane removed. In some places there is an increase in fibrous tissue, especially around the opening made in the canine fossa.

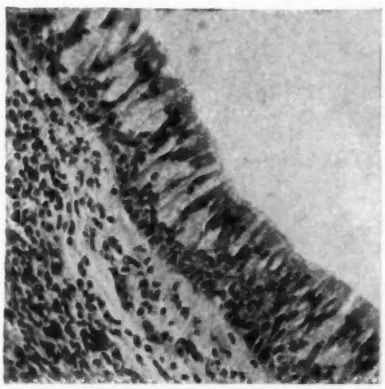

FIG. 1—Normal human antral lining, showing ciliated columnar epithelium with many goblet cells.

The method of repair is the same in all cases, but certain factors tend to cause some variation in this repair. Some individuals are prone to form excessive granulation tissue during the healing process. The presence of infection in neighboring sinuses or reinfection of the new lining may alter the course of repair. Also the general health of the patient is an important factor. But the fact remains that the mucous membrane does regenerate.

This slide shows normal mucous membrane from the antrum with its ciliated epithelium containing goblet cells and the delicate connective tissue stroma containing some lymphocytes and a few mucous glands.

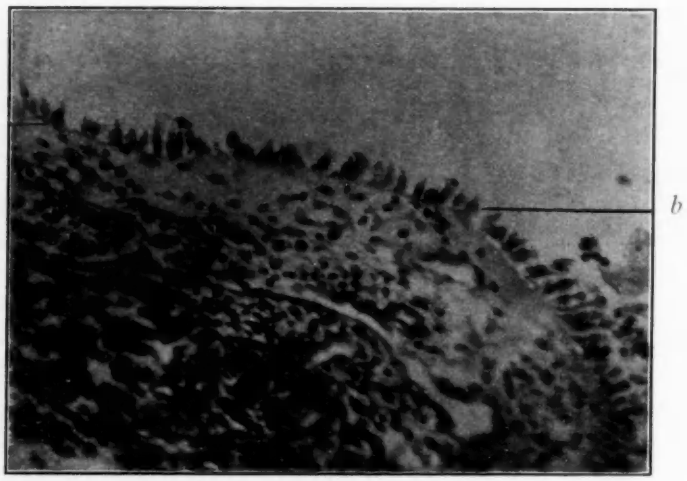

FIG. 2—Sixteen day specimen from a human antrum, showing young regenerating epithelial cells growing on the surface of granulation tissue. Low power view. *a* indicates the basement membrane; *b*, epithelium.

We do not know exactly when reformation is complete. There is probably considerable variation in this respect. But we do know that regeneration begins very early, as demonstrated by Figs. 2 and 3.

Here sixteen days after operation, we see young regenerating epithelial cells, growing on the surface of granulation tissue. Macroscopically the whole antrum cavity was lined by a very thin velvety appearing material which was grey in color, and quite moist and friable.

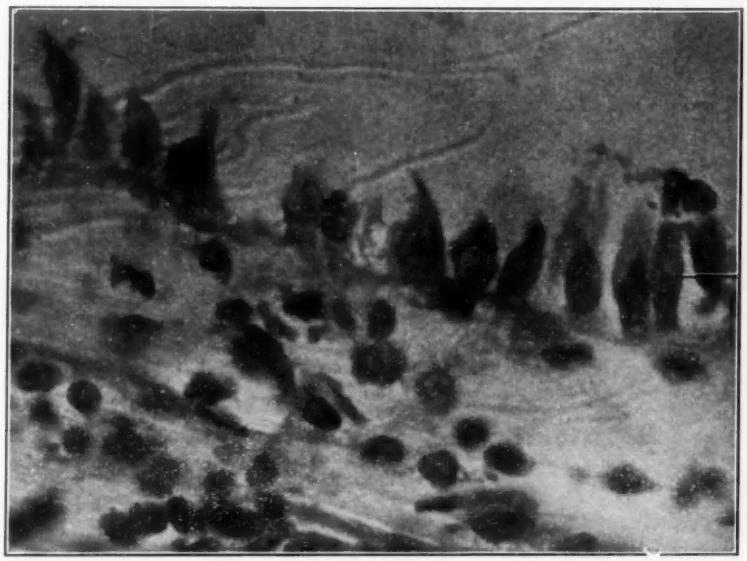

FIG. 3—Sixteen day specimen from a human antrum, showing young regenerating epithelial cells (*a*) growing on the surface of granulation tissue. High power view.

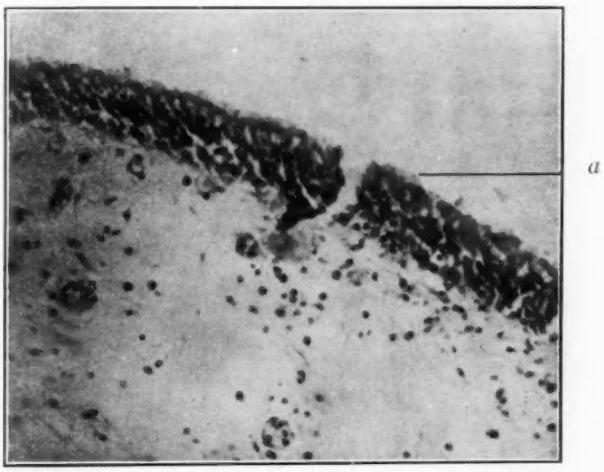

FIG. 4—Six month specimen from a human frontal sinus, showing regenerated ciliated columnar epithelium (*a*). Low power view.

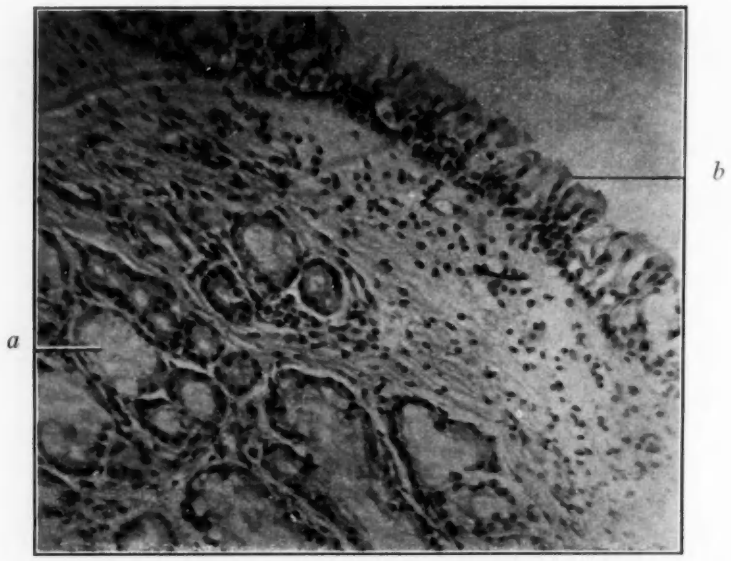

FIG. 5—Seven month specimen from a human antrum, showing regenerated ciliated columnar epithelium (*b*) with glands (*a*) in the stroma. Low power view.

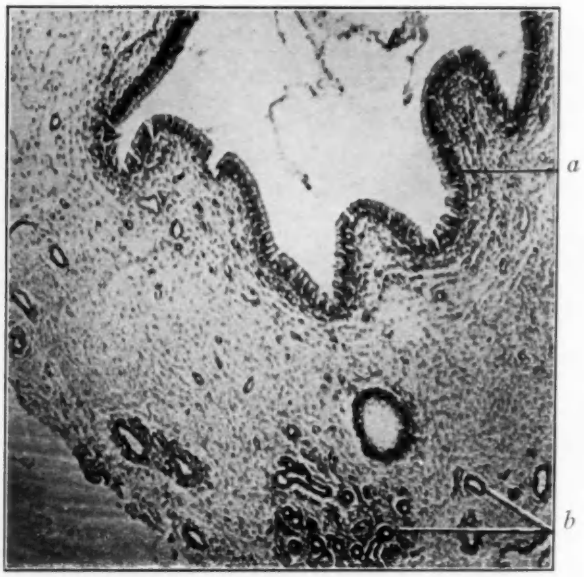

FIG. 6—Two year specimen from a human antrum, showing regenerated ciliated columnar epithelium (*a*) with glands (*b*) in the stroma. Low power view.

Although this is from frontal sinus it shows well-formed mucous membrane lining six months after operation. The surface is covered by ciliated columnar epithelium. To the eye it appeared to be completely relined with normal mucous membrane.

Seven months after operation this antrum shows regenerated ciliated columnar epithelium, with numerous glands in the stroma. To the eye the whole surface appeared to be covered by normal mucous membrane. A partial partition had formed, and this also appeared to be covered by normal mucous membrane.

This antrum was reopened because of pain, two years after the first operation had been performed. The lining appeared to be perfectly normal and microscopic examination substantiates the gross appearance. The epithelial layer rests on a thin basement membrane. It contains many distended goblet cells and it is completely covered with cilia. The stroma is very thin and contains a few lymphocytes, and a considerable number of glands.

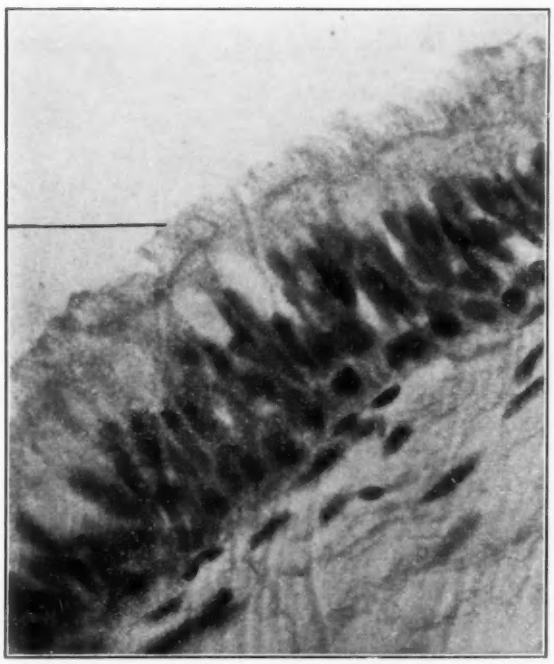

FIG. 7—Regenerated columnar epithelium from a human antrum, showing cilia. High power view. *a* indicates ciliated epithelium.

This is a high-power view of regenerated columnar epithelium showing cilia.

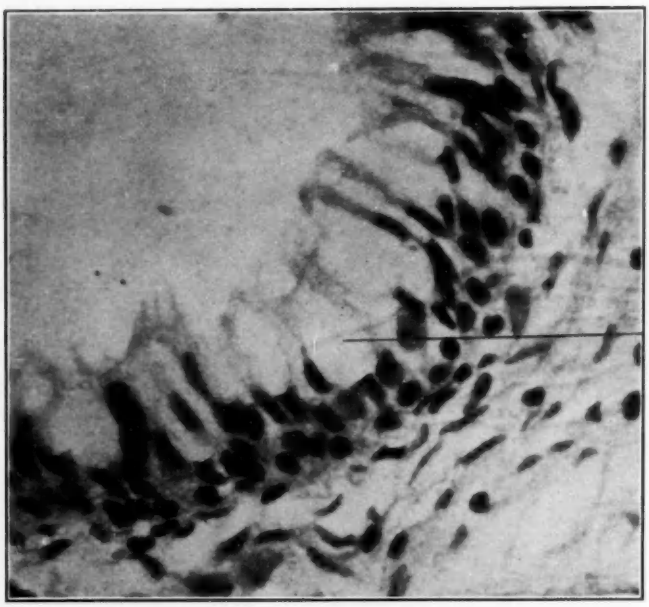

FIG. 8—Regenerated columnar epithelium from a human antrum, showing goblet cells (*a*). High power view.

This is a high-power view of a regenerated columnar epithelium showing goblet cells. The presence of goblet cells is not an evidence of mucoid degeneration but is evidence of the presence of a perfectly normal physiological process. The number of goblet cells varies tremendously according to the activity of the membrane.

This slide shows regenerated ciliated columnar epithelium from six antra which previously had had the radical operation. There is not sufficient time to demonstrate every case, but these are sufficient to show that mucous membrane does regenerate in the antrum after complete removal of the lining.

Slide 10 — I put on this slide to show that histiocytes and reticulo-endothelium are present in the new lining. Many of the cells you see are histiocytes and endothelial cells.

Criticism has been made, that although the regenerating mucous membrane may appear normal it may not have regained its normal function. However, little is really known about the normal function. The chief functional elements are the surface

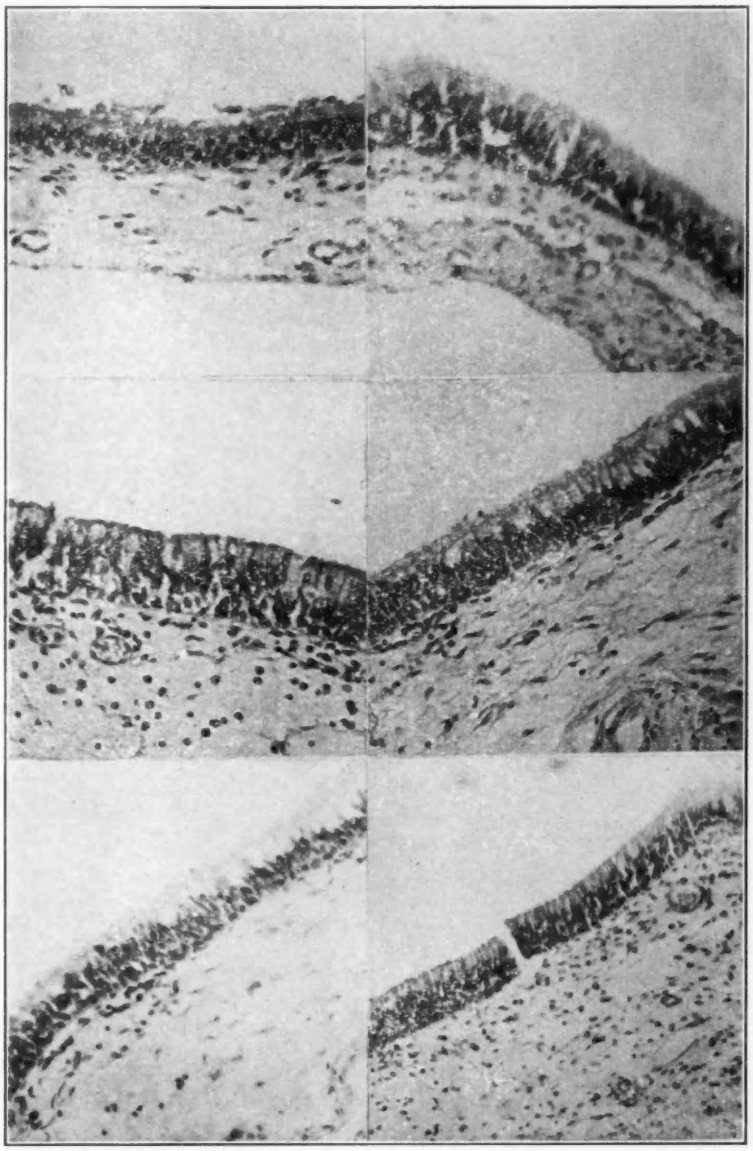

FIG. 9 — Specimens from human antrums, showing regenerated ciliated columnar epithelium. Low power view. These specimens were removed at various intervals following the radical operation, the longest period being nine years.

epithelium with its goblet cells and cilia and the stroma with its mucous glands, histiocytes and reticulo-endothelial system. Nasal mucosa has the power locally to form specific agglutinins and antibodies, due to the presence of its reticulo-endothelial system. It may be that the mucosa of the antrum has the same power and when overcome by disease it loses this power. The presence of a surface covering of ciliated columnar epithelium with goblet cells together with mucous glands, histiocytes, and a reticulo-endothelial system in the new lining would lead one to believe that the new lining would have functions similar to those of the old lining. An increase in the amount of fibrous tissue in the new lining may be beneficial because it binds the membrane down to bone, preventing the rapid swelling and edema which comes so readily with infection of the mucosa. One knows that the functional elements of the mucous membrane lining regenerate and when these appear normal the logical assumption is that their function is normal.

III. REFORMATION OF THE MUCOUS MEMBRANE OF THE MAXILLARY SINUS

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BOSTON, MASS.

TO BEGIN this brief paper it would be well, it seems to me, to ask this question: Does the mucous membrane of the maxillary sinus reform following radical operation? Also, is it not true that we may have a perfect regeneration of mucous membrane in the maxillary sinus following the radical operation? And further, what are the interesting features regarding origin, appearance and time relating to the structural changes in the antrum following operation?

It seems to me that the best way to cover the above statements and also to keep fairly within the time allowed will be to show on the screen the reproduction of microscopic slides, more or less familiar to some of you, of work done on dogs by Dr. Mosher's men, some time ago.

Dog No. 1: At autopsy, one month after operation, the opening through the maxilla was found covered by a layer of somewhat resistant tissue nearly an eighth of an inch thick. A section of this area and of a portion of the new lining surrounding the bony opening showed, in its early stages, new fibroblastic tissue well arranged. On the surface of this newly-formed tissue was a thick layer of red cells into which the new fibroblastic tissue and blood capillaries from below were making their way preparatory to replacing it. On the surface of this layer of red cells was a delicate layer of columnar ciliated epithelium with goblet cells already in evidence, but which had not as yet completely bridged the gap. Bone spicules also were appearing at the margin of the opening.

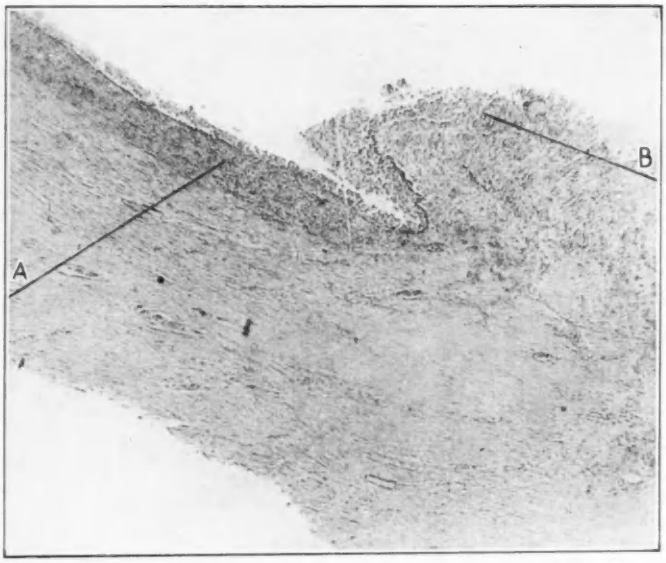

FIG. 1—(Dog 1)—Section of the antral lining, showing a deep layer of organizing red blood cells covered by new ciliated columnar epithelium not as yet completely extended across the canine fossa opening. A, indicates the deep layer of organizing red blood cells; and B, the end of the new epithelium.

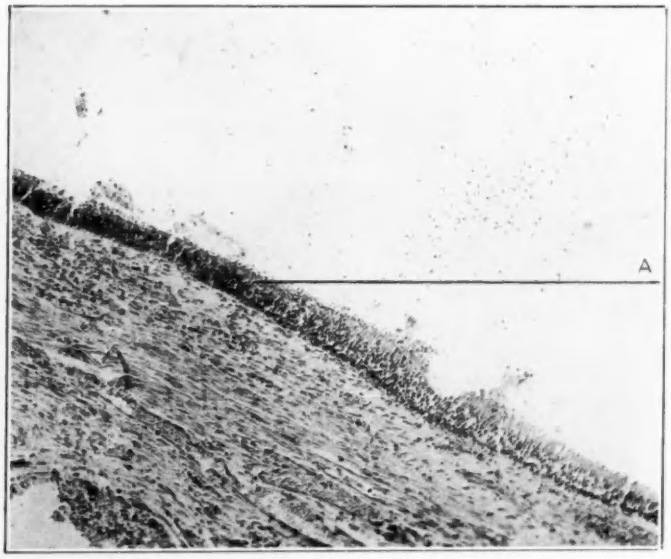

FIG. 2—(Dog 1)—Section showing the point at which the new epithelium has grown out from the old, the dark being the old and the light, the new. The latter is seen to be a loosely-constructed layer at this time. A, indicates the beginning of the new epithelium.

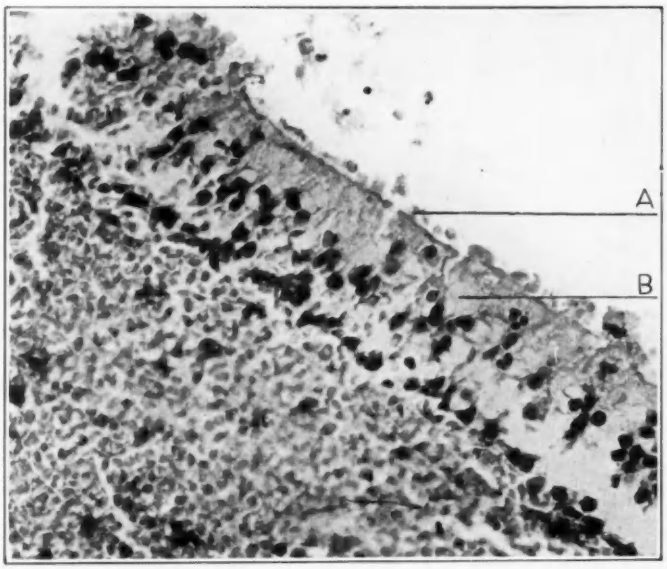

FIG. 3—(Dog 1)—High power field, showing new columnar ciliated epithelium with large goblet cells growing over a mass of red blood cells. A, indicates the cilia; and B, the large goblet cells.

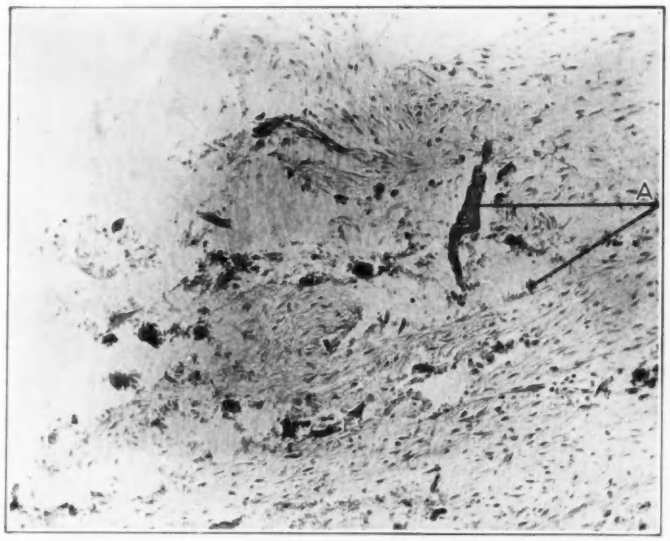

FIG. 4—(Dog 1)—Section showing new bone spicules scattered promiscuously in the new fibroblastic tissue filling in the canine fossa opening. A, indicates the bone spicules.

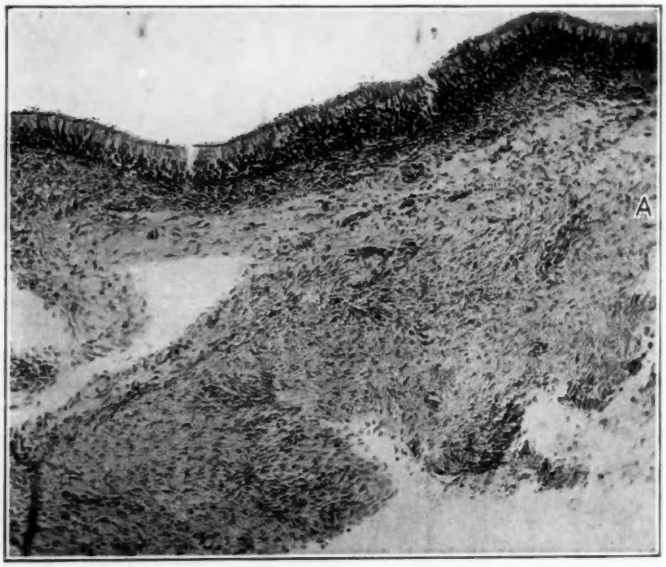

FIG. 5—(Dog 2)—Section showing perfectly-formed ciliated columnar epithelium with large goblet cells over a thick layer of new fibrous tissue. No glands have as yet appeared; the red cells have vanished. A, indicates the fibroblastic tissue.

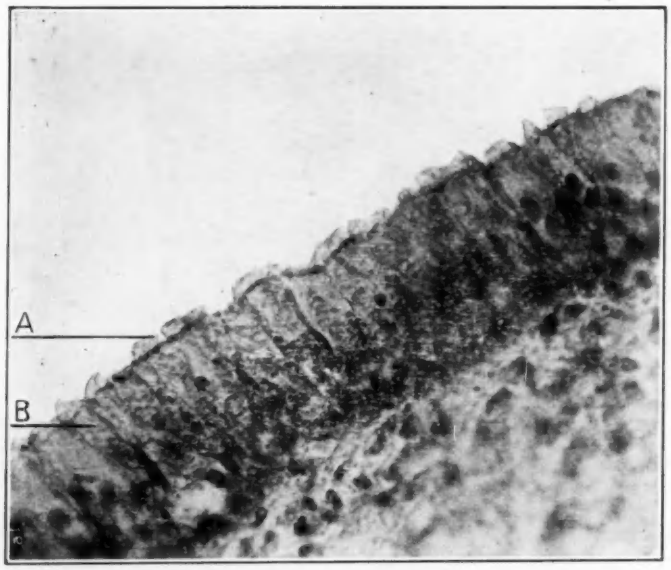

FIG. 6—(Dog 2)—High power field, showing completely regenerated ciliated columnar epithelium with a great number of large goblet cells. A, indicates the cilia; and B, the goblet cell.

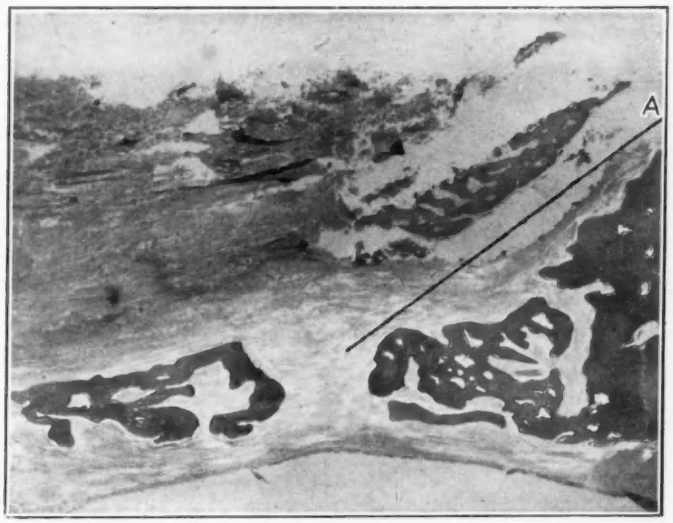

FIG. 7—(Dog 2)—Section showing regenerated bone nearly closing the canine fossa opening. A, indicates a 1.5 mm. area not yet ossified.

Dog No. 2: Autopsy three months after operation. The canine fossa opening was nearly closed by a deposit of bone in the fibroblastic tissue. The new lining of the antrum was made up of well-developed fibroblastic tissue covered with a perfectly-formed columnar ciliated epithelium which showed large goblet cells. There was no vestige of gland regeneration.

Dog No. 3: Autopsy five months after operation. Here the canine fossa opening was closed completely with bone. The epithelium was a perfectly-developed columnar ciliated type with large goblet cells. There was typical gland regeneration. A few collections of lymph cells resembling lymphatic nodules were seen. The subepithelial tissue was vascular. The fibroblastic tissue was now firmer, its cells approaching the adult type and the whole antral lining was assuming the appearance of a normal mucoperiosteum.

Slide number ten is the last contribution I have to offer from our canine friends. There is one more slide to show. The specimen was sent to me by Dr. McGregor soon after we had worked on the dogs. The specimen was taken from one of Dr. Goldsmith's patients at the third proposed radical operation which was begun because of pain. The specimen shows as nearly a normal antral lining as one would wish to see.

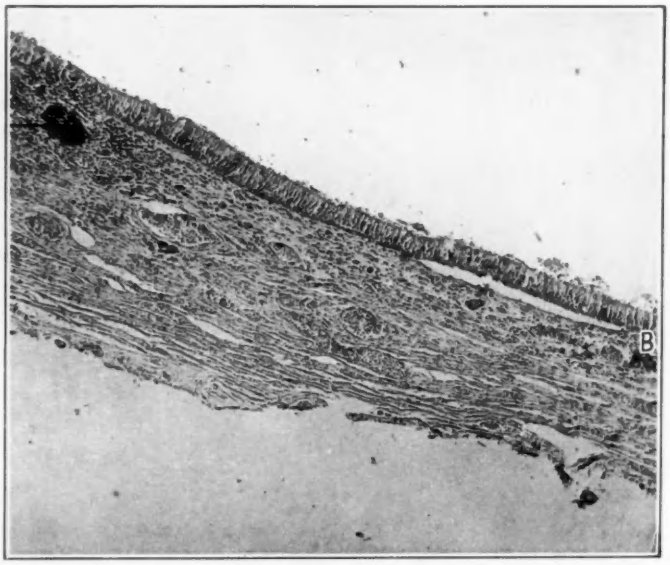

FIG. 8—(Dog 3)—Section showing regenerated antrum lining with ciliated columnar epithelium, masses of lymphocytes and gland structures. A, indicates the collection of lymphocytes; and B, the glands.

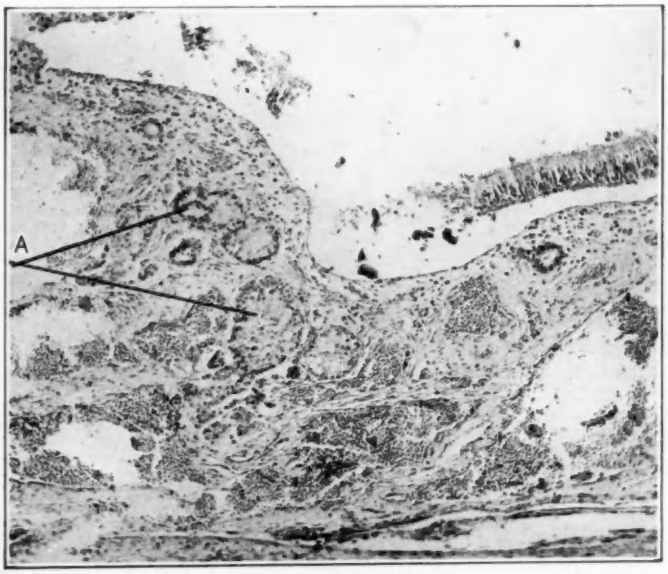

FIG. 9—(Dog 3)—Section showing gland regeneration. A, indicates glands.

FIG. 10—(Dog 3)—Section showing regenerated muco-periosteum with, among other glands, one growing down from the epithelium. A, indicates a glandular outgrowth from the epithelium.

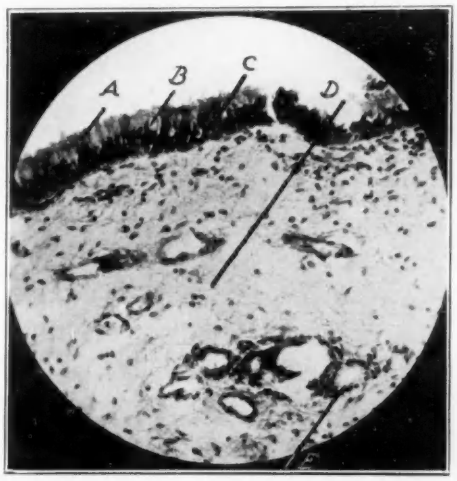

FIG. 11—A, The cornstalk cilia; B, Goblet cells; C, Columnar cells; D, The thin, normally-constructed periosteal layer; E, The glands with their secreting cells.

IV. AN EXPERIMENTAL AND CLINICAL STUDY OF HISTIOCYTES IN ACUTE AND CHRONIC INFLAMMATION OF THE ACCESSORY SINUSES

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PORTLAND, ORE.

SINCERE THANKS are due the Research Committee of the Council of the American Academy of Ophthalmology and Otolaryngology for its generous grant of funds to continue animal work and histologic study of histiocytes during the academic year 1931-32, along lines reported before this Academy one year ago. Many hundreds of serial sections were prepared from cat frontals and from selected human maxillary membranes. Detailed report of each type of experiment will be found herewith. Numerous agglutinative substances capable of arresting ciliary action were first tried, then certain alkaline earths and chlorids, and a limited number of human experiments based on the latter group were made.

In cat material the histiocytes show trypan blue granules, due to vital staining. In human material the histiocytes show no granules, because vital staining is impossible; but these cells are differentiated by Maximow's stain, by their grayish cytoplasm and their vacuoles, together with the characteristic appearance of the nucleus and the triangular or crescentic shape of the cell. Eosinophils and basophils show their characteristic granules in both human and cat preparations, staining much as in blood smears treated with Wright's stain. All cat material was secured by needle puncture of the readily accessible frontal sinus, subsequent subcutaneous injection of 1 per cent trypan

* Research authorized and financed by The American Academy of Ophthalmology and Otolaryngology, in the Departments of Otolaryngology and Anatomy of the University of Oregon Medical School, Portland. Presented at the Montreal meeting of the American Academy, September 19, 1932.

blue, and fixation of the acutely inflamed membrane after forty-eight to seventy-two hours, as heretofore described. Injected substances were, of course, autoclaved to prevent added infection.†

Cat Material

After injection of castile soap jelly, the entire mucosa is edematous, somewhat injected, and filled with pus cells, which have penetrated the epithelium until the latter is almost unrecognizable.

Thin glucose solution showed similar epithelial destruction, with mucosa filled with pus cells, but a few histiocytes in the deep part of the tunica propria.

A fifty per cent emulsion of cocoanut oil with agar showed some injury to the free ends of the epithelium, clogging of cilia, and considerable mucous secretion. The tunica propria was somewhat injected, otherwise nearly normal. Histiocytes in normal numbers, but numerous leukocytes; no very marked effect produced.

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† Note on staining for histiocytes in human material.

Numerous requests for the technique of the so-called Maximow stain, with counterstain of hematoxylin, have been received. More details may be secured from McClung's Microscopical Technique.

Zenker-formal fixation is preferred; however, Susa gives excellent results. Section should be from 6 to 8 microns.

The Delafield hematoxylin, not artificially ripened, should be about six weeks old. Make a very dilute solution of it by adding 2 to 6 drops to 100 cc. distilled water; stain 24 hours. Wash in distilled water 24 hours. Result: chromatin is blue, cytoplasm either colorless or very light gray.

Place in azure II-eosin for 24 hours. Prepare as follows:

Stock Solution A—
Eosin, water sol., yellowish (W.G.) ..... 0.5 gm.
Distilled water .....500. cc.

Stock Solution B—
Azure II ..... 0.5 gm.
Distilled water .....500. cc.

The Stain:
Solution A, 10 to 12 cc. according to material
Solution B, 8 to 10 cc. according to material
Distilled water, 100 cc.

For the Susa fixed material used, best results were obtained by using 13 cc. of Solution A and 8 cc. of Solution B. Mix the eosin solution and water first, then add the azure solution. A noticeable precipitate should not form for several hours. Leave slides upright in the stain 24 hours. Transfer from stain to 95 per cent alcohol, then 100 per cent to differentiate and dehydrate. Finish dehydration in second 100 per cent alcohol, clear and mount. The differentiation takes place rapidly in 95 per cent alcohol, more slowly in 100 per cent.

Chromatin of the nuclei is dark blue, nucleoli are purple, and leukocyte granules are differentially stained in colors corresponding to those given by Giemsa.

Jelly of chondrus crispus (which contains a good deal of salt) showed some parts of the membrane injected with blood and considerably thickened, while the tunica propria was full of leukocytes, with a few histiocytes. Elsewhere the mucosa was completely destroyed, with a great mass of pus in the sinus cavity surrounding shreds of the moss.

Mucilage of tragacanth (without the usual preservative addition) caused little injection or thickening of the mucosa. The epithelium was somewhat damaged, with clumping and occasional destruction of cilia. There was considerable mucous secretion with enlargement of goblet cells. Pus cells were frequent in the tunica propria, migrating through the epithelium, and in the gum out in the sinus cavity.

Thick glucose left the sinus membrane of normal thickness, neither edematous nor injected. The epithelium was fairly normal in appearance, but the cilia were badly gummed together. Goblet cells were numerous and large, with an abnormal amount of mucous secretion. Some leukocytes and about the normal number of histiocytes were found in the tunica propria. The chief effect was apparently clumping and apparent stasis of cilia, and production of a large amount of mucous secretion.

Milk of magnesia produced very great numbers of histiocytes, but the epithelium was largely destroyed.

A repetition of the experiment with chondrus crispus, permitting a longer time to elapse, gave much the same result as before. The epithelium became so infiltrated with leukocytes as to be almost unrecognizable. Many lymph follicles were found in the tunica propria, pressing up on the epithelium. It was hard to recognize the histiocytes, but they were not numerous.

Petrolagar with ten per cent glycerine left the epithelium intact. Some lymph follicles were found under the epithelial layer, wherein the goblet cells were much swollen. Numerous histiocytes were present.

Calcium hydroxide five per cent, emulsified with one-half per cent agar, showed the cilia destroyed and much epithelial damage, with marked migration of leukocytes through the epithelium. The tunica propria showed many polymorphonuclear cells, some lymphocytes and plasma cells, and large numbers of histiocytes. The latter were scattered through the edematous tunica propria, appearing to be migrating toward the epithelium.

Calcium lactate one per cent with gelatin one per cent caused bad destruction of the epithelium in spots. The mucosa was thick, edematous, and full of various types of cells, including numerous lymphocytes, some polymorphonuclears, some plasma cells, and very numerous histiocytes, especially just beneath the epithelium. In spots where the epithelial cells are nearly destroyed, the histiocytes form a continuous layer at the base of the epithelium (Fig. 1).

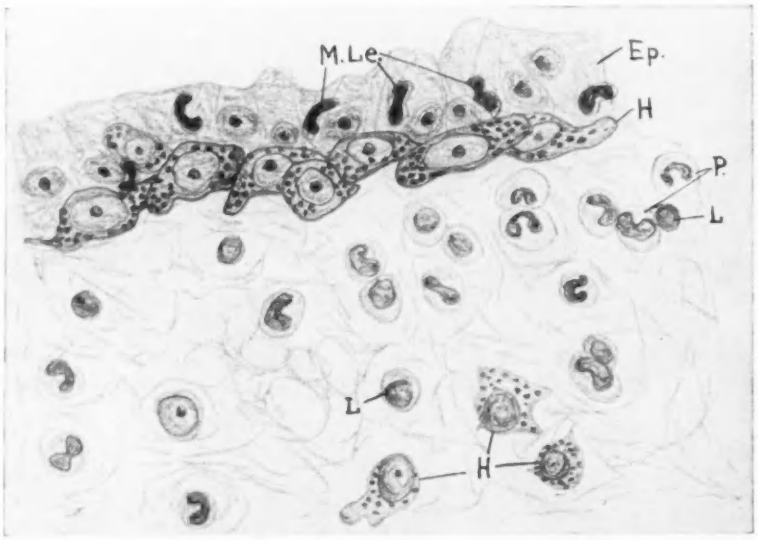

FIG. 1—Frontal sinus membrane, cat. Sinus had been injected with calcium lactate 1% in 1% gelatin. The animal then received subcutaneous injection of 0.5% trypan blue in normal saline. A barrier of histiocytes is clearly seen under the damaged epithelium. Fixed after 48 hours; stain, hematoxylin-eosin-azur. Camera lucida drawing, 10 oc. 90 obj. (oil immersion) (Larsell). *Ep.*, epithelial cells; *M.Le.*, migrating leukocytes; *H.*, histiocytes containing trypan blue granules; *P.*, polymorphonuclears; *L.*, lymphocytes.

Sodium phosphate one per cent with two per cent gelatin gave a relatively normal mucosa, somewhat swollen in spots, but not very edematous. The cilia remained well shown. A somewhat higher number of histiocytes than usual occurred in the tunica propria. The edematous areas contained some polymorphonuclears and a few plasma cells. A control animal in which plain gelatin was used died from infection, and the tissues were not prepared.

Scarlet red five per cent with oxyquinoline sulphate two per cent, as used for stimulation of skin grafted areas, caused mucosal edema and epithelial damage, with patchy loss of cilia. Leukocytic migration was occurring through the epithelial layer, and the tunica propria was full of pus cells. Very few histiocytes were present.

Dichloramin two per cent in chlorcosane was responsible only for unusually large goblet cells, other findings being normal. This experiment should be repeated, as double penetration occurred, with but little oil in the small frontal, while epidural hemorrhage caused death of the animal.

One animal was injected on the right side with a virulent hemolytic upper respiratory streptococcus in semiliquid gelatin culture. The left side was studied as a control. Two days after the sinus had been infected, one cc. of vaccine made up from the same organism was injected intradermally after the method recently introduced by Corbus. Another injection of vaccine was given two days later, and after two more days trypan blue was injected and the material was fixed. The infected sinus showed slight hyperemia when dissected out. In section the membrane was hyperemic and edematous, the epithelium tall, with many mucous cells. The subepithelial tissue contained many histiocytes and plasma cells; and in spots numerous lymphocytes and leukocytes, with great thickening of the mucosa and epithelial layer. At such areas, the epithelial cells were elongated and devoid of cilia, and the layer of mucous secretion normally covering the epithlium was absent. Apparently the infection had passed at these points into the underlying tissue. Leukocytes in considerable numbers were migrating through the epithelium, and masses of pus were found in some parts of the sinus cavity. The fibroblasts were more frequent than normally in the tunica propria. At other points of the infected sinus transitional stages from the marked infection just described occurred, grading into nearly normal thin ciliated mucosa with few goblet cells. At such spots, the tunica propria seemed to contain rather more than the normal number of histiocytes.

The control (left) side was entirely normal except for a very definite increase in the number of histiocytes, also a few plasma cells. These findings, apparently confirmatory of the assertion of Corbus that intradermal vaccines stimulate histio-

cytic defense wherever inflammatory lesions are found, should, of course, receive numerous experimental confirmations before they can be accepted.

Human Material

Individuals with apparently equal involvement, clinically and radiologically, were subjected to various types of local treatment; and after the lapse of a certain time, varying with the needs of each case, the membranes were carefully removed at radical operation, fixed immediately, and examined under the eosin-azur differential stain for histiocytes and other elements.

Case 12707, used to clarify points of technique, had areas of broken-down epithelium with subjacent edema; elsewhere the usual tall columnar cells with numerous goblet cells occurred, and the edema was much less, and the tunica propria fibrous. Under the broken areas were numerous plasma cells, some round cells,

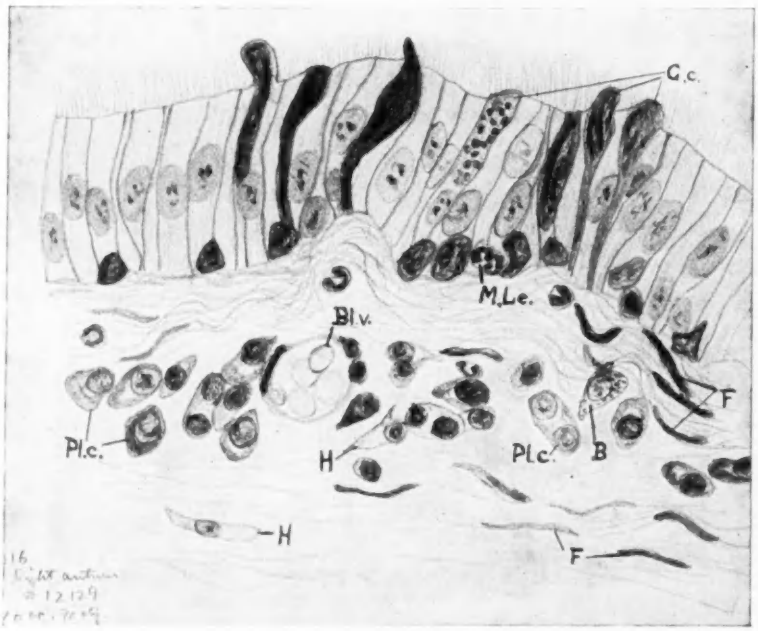

FIG. 2—Maxillary antrum membrane, human, case 12129, removed at radical operation. Plasma cells predominate in this very fibrous membrane, and histiocytes are few. Hematoxylin-eosin-azur. Camera lucida drawing, 10 oc. 90 obj. (oil immersion) (Larsell). *G.c.*, Goblet cells; *M.Le.*, Migrating leukocytes; *Bl.v.*, Blood vessel; *B.*, Basophil; *H.*, Histiocytes, showing white vacuoles in gray cytoplasm; *Pl.c.*, Plasma cells; *F.*, Fibroblasts.

and some histiocytes. Under intact epithelium, lymphocytes predominate, occasionally as small follicles. There are also numerous fibroblasts and occasional basophil cells.

Case 12129 presented an intact epithelium with no migration of leukocytes and little edema. The tunica propria was fibrous but rather thin, and filled with very great numbers of plasma cells. Comparatively few histiocytes were found. These plasma cells formed a definite, though not compact layer beneath the epithelium (Fig. 2).

FIG. 3—Maxillary antrum membrane, human, case 11432, removed at radical operation. Marked edema of the tunica propria and very great number of eosinophils. Hematoxylin-eosin-azur. Camera lucida drawing, 10 oc. 40 obj. (Larsell). *Gc.*, Goblet cells; *M.Le.*, Migrating Leukocytes; *H.*, Histiocytes; *B.*, Basophils; *E.*, Eosinophils; *Pl.c.*, Plasma cells; *L.*, Lymphocytes; *Bl.v.*, Blood vessel.

Case 11432, left side, had received a few washings with normal saline solution. Here the epithelium was tall, and cilia absent in patches, elsewhere clumped, occasionally normal. Very many mucous cells and migrating leukocytes were found. The tunica propria was very edematous, containing numerous lymphocytes, many plasma cells, and an occasional mast cell. Very many eosinophiles, some histiocytes and many fibroblasts completed the picture (Fig. 3).

The right side, clinically identical, received one 6 cc. injection of milk of magnesia. No epithelium was visible in these sections; there was marked hemorrhage. The tunica propria was dense and fibrous and the predominant cells were fibroblasts. It is not unlikely that the radiologic and clinical similarity of these two sides were due to the thick fibrous repair on the right, and that merely the hemorrhage may be laid to the magnesia.

The right antrum of D. H. was injected with milk of magnesia once or twice a week for two months. This membrane was markedly edematous. Lymphocytes were very numerous about the blood vessels. Plasma cells were numerous, especially about the mucous glands in the deep part of the mucosa. Very numerous eosinophiles were found near blood vessels and in the tunica propria where epithelium was missing. Histiocytes were fairly numerous, and some fibroblasts were scattered through the tunica propria. Certain pigmented cells were evidently degenerative. The tall epithelium showed almost every other cell a goblet cell, indicating degeneration; but most of the epithelial cells were still ciliated. Here and there the epithelium was lost, and much mucous secretion was present. A few almost normal patches were found.

Very different was the left antrum in the same case, washed for two months with normal saline. This was much more edematous, with large spaces in the tunica propria waterlogged by fluid. There was marked infiltration by lymphocytes, with patches of lymphoid tissue. The epithelium was markedly degenerated in spots, with cilia absent from considerable areas; elsewhere normal, save for unusual quantities of secretion, with half the cells goblets. Where the epithelium was degenerated, very little secretion was seen. Plasma cells were more numerous than other types, save for the perivascular lymphocytes in the tunica propria. Few histiocytes were to be found, and no eosino-

philes. This raises the question whether the marked eosinophilia of the right side was due to the long exposure to the milk of magnesia, by chemotaxis, or by dehydration changes in the mucosa. Certainly there was a striking absence of eosinophiles in the left side, where normal saline was used.

Recapitulation

In the cat, destructive changes in the mucosa of the frontal sinus, without any apparent increase in the histiocytic defense, were produced by soap jelly, thin and thick glucose, cocoanut oil with agar, chondrus jelly, mucilage of tragacanth, and scarlet red with oxyquinoline sulphate.

Certain kations produced notable increase of histiocytes in the membrane, in spite of loss of cilia and more or less epithelial damage. These substances included calcium hydroxide five per cent in one-half per cent agar; calcium lactate one per cent in one per cent gelatin; and sodium phosphate one per cent in gelatin two per cent. Especially interesting is the calcium lactate effect: the epithelium badly destroyed in spots, under which a continuous layer of histiocytes has formed (Fig. 1). Numerous lymphocytes, some plasma cells, and some polymorphonuclears are found in the edematous stroma.

Human material was secured by radical antrum operation on certain individuals with apparently equal involvement on each side, both clinically and radiologically. This material was divisible into three histologic classes:

1. fibrous repair, associated with which plasma cells were observed in large numbers (Fig. 2);
2. an apparently allergic type, with much edema and many eosinophils, and slightly greater numbers of histiocytes and plasma cells than normal (Fig. 3); and
3. mixed types, varying apparently with the location, conformation and healing of chronic inflammatory lesions.

The time factor in human material was introduced by washing one antrum with milk of magnesia, the other with normal saline, for two months once or twice weekly. Marked edema, with very numerous perivascular eosinophiles, fairly numerous histiocytes and some fibroblasts, characterized the "magnesia" side. On the "saline" side, edema was excessive, with marked

lymphoid infiltration and loss of cilia and of epithelial patches. Numerous plasma cells were found about mucous glands, with but few histiocytes and no eosinophiles.

Intradermal immunization (after Corbus) was attempted on a cat which had been infected in one frontal by a virulent gelatin culture of hemolytic streptococcus. Both the normal and affected side showed many more histiocytes than normal, seeming to corroborate Corbus's theory that intradermal vaccination activates histiocyte defense at points of inflammation throughout the body.

Conclusions

Subject to further experimental confirmation on human subjects, it would seem that local use of solutions or suspensions of alkaline-earth salts or hydroxides (calcium, magnesium) favors the mobilization of histiocytes in acute inflammation. Oily and colloid substances are destructive of epithelium and favor infection by impairing ciliary action, necessitating invasion of the subepithelial stroma by polymorphonuclear cells without any increase in the number of histiocytes. The chlorides seem to favor edematous changes with marked increase of lymphocytes. An increase of plasma cells seems to accompany long-standing fibrous changes.

The Department of Radiology and the Department of Anatomy are now engaged in a series of experiments on cats to provoke chronic inflammations of the frontal sinuses, upon which the X-ray will later be used.

Further experiment, to determine the influence of acid, saline and alkaline diet, to check further the merits of intradermal vaccine treatment, and to decide on the function of the plasma cell, is necessary and will be undertaken when necessary funds for technical help and material are available.

V. A STUDY OF NASAL MUCOUS MEMBRANE IN HANGING-DROP TISSUE CULTURES

P. J. Ross, M.D.

BOSTON, MASS.

TISSUE CULTURE methods have been used in the study of normal and pathological tissues for the past twenty years. In spite of this and the fact that the nose and the accessory sinuses are able to supply tissue materials in considerable quantities, these methods have not been used to any great extent in the study of the nasal mucous membrane.

The purpose of this paper is briefly to record some of my observations regarding the behavior of human nasal mucous membrane in tissue cultures. This work was done in the laboratory of Dr. Harris P. Mosher at the Massachusetts Eye and Ear Infirmary.

Culture Methods — The hanging-drop method was used in this study because of its relatively simple technique and because of the difficulty of obtaining bacteria-free tissues. The culture medium was made of equal parts of a solution containing glucose and inorganic salts in the proportions found in blood plasma, and an embryonic extract, the embryonic extract being a saline extract of fresh umbilical cord. Previous workers have used an extract of animal embryos. Since these were not available for this study, I found that fresh umbilical cord gave a suitable embryonic extract. The hydrogen ion concentration of the medium was adjusted to pH 7.4 and the media then passed through a bacterial filter before use. Selected fragments of tissue were washed in several changes of this medium and then sealed in the hanging-drop slide. The slides were then inverted and incubated at body temperature. The medium was changed every twenty-four hours.

Tissue Materials — The tissues used in this study were obtained from the clinics and the operating rooms of the Massachusetts Eye and Ear Infirmary.

The first cultures were made from sinus membrane removed at operation. These tissues all showed chronic inflammatory changes and were usually so badly traumatized and macerated with the purulent contents of the sinus at operation that satisfactory fragments for culture were obtained with difficulty. In spite of this, an active ciliated epithelium was found in all specimens removed from the maxillary sinus, regardless of the chronic inflammatory changes found in the sinus at operation. Specimens from the diseased frontal sinus did not as a rule show an active ciliated epithelium. In these cases the membrane was found to be replaced by granulation tissue arising from adjacent diseased bone.

Tissue cultures from the nasal cavity proper were not satisfactory because the epithelium in most instances had undergone a metaplasia to the stratified type.

Nasal polypi gave the most uniformly satisfactory growth. Aside from the edema and the smaller blood supply the translucent nasal polyp may be said to be histologically the same as sinus membrane. It is covered with a normal-looking epithelium and the stroma does not differ greatly in its cellular makeup from that of normal sinus membrane. Polypi are easily obtained, are easily washed and present a smooth curved surface for the selection of fragments for culture.

Observations — The first thing to engage my interest was the identification of the cells, and the order of their migration from the edge of the tissue outward along the cover slip. Although vital stains were used, and fixed and stained preparations were studied, it was found necessary to study and to be able to identify the various cells in their unstained living state. The first cells to appear at the edge of the tissue are the polymorphonuclear leukocytes. They move by ameboid motion outward into the media and disintegrate in twenty-four to forty-eight hours. The cytoplasmic granules are in constant motion until the cell dies. These cells are followed by the histiocytes or large mononuclears of the blood and tissues which appear in large numbers in from twelve to twenty-four hours. The histiocytes become rounded in the fluid media but adhere to the coverslip by long processes. These cells at first have a relatively clear cytoplasm that soon becomes filled with granules and vacuoles which stain quickly when vital dyes are added to the media.

Fibroblasts appear in twenty-four to forty-eight hours and grow outward into the preceding cells, giving by their spindle shape a striated appearance to the growth. This appearance of the fibroblasts, however, gives no characteristic structure to the growth and no definite rearrangement of the cells was noticed in any of the cultures.

The epithelial cells were the most interesting of the cells studied. Ciliated and non-ciliated forms were identified. The epithelium is very loosely attached to the basement membrane and is seen in the media both as living and dead cells, either singly or in large clumps. The living cells quickly become rounded in the fluid media, whereas the dead cells retain their compressed elongated forms. The non-ciliated cells are probably immature or replacement cells that have not developed cilia.

No goblet cells were recognized as such; however, after forty-eight hours' incubation many of the epithelial cells developed a clear vesicular projection which was attached adjacent to the cilia. This may be the so-called goblet cell formation which in the isolated cell in the culture distorts the cell by a clear rounded projection, whereas the cell in position on the basement membrane, being supported on all sides by other cells, is not so distorted and this vesicle or vacuole appears to be a large clear space in the free end of the cell. These vesicles were attached to both ciliated and non-ciliated cells and I was unable to determine whether or not it was a degenerative process, although I am inclined to think that it is due to an alteration of the chemistry of the media.

The Cilia — The cilia are attached to a circular plate on the surface of the cell which is a thickening of the limiting cellular membrane. In the rounded cell in culture this plate forms a smaller arc than the remainder of the circumference and may be likened to the cornea of the eye in its relation to the rest of the globe. The cilia are embedded in this plate by a rounded enlargement of their attached ends and this enlargement is the axis of motion of the cilia, being present in both dead and living cells.

An attempt was made to determine the mode of development of the cilia. The non-ciliated epithelial cells have a nucleus and cytoplasmic structure similar to the ciliated forms and after some experience are easily recognized as epithelial cells. After

from four to five days of incubation many of these non-ciliated cells developed a small tuft of from three to ten or more extremely fine fibrils. These were about twice the length of the ordinary cilia and were just visible under the oil immersion objective. At the point of their attachment there was a slight bulging but no thickening of the cell membrane. These fibrils were not motile and a complete development into true cilia did not occur.

The Presence of Bacteria — The difficulty of obtaining bacteria-free tissues was mentioned. Approximately one-third of all the cultures were killed by bacterial overgrowth in the first twenty-four hours. It is possible by repeated washing of the polypi and fragments of the membrane to obtain bacteria-free cultures. This fact leads me to feel that actual bacterial invasion of the nasal mucous membrane is uncommon.

In many of the cultures the presence of bacteria did not interfere with tissue growth. It was in these cultures that an interesting phenomenon occurred. The histiocytes showed a marked attraction for the bacteria, ingesting many, while many bacteria were attached to the surface of these cells and large numbers were present in the adjacent media. These histiocytes were finally overcome and quickly disintegrated. It was also interesting to note that the epithelial cells were not attacked by the organisms, and in all instances seemed to repel them. The media would be clear and free of bacteria for two or three microns about the epithelial cells. This was true of both dead and living cells and in cultures dead for two or three weeks the majority of remaining cells would be intact epithelial cells. In no instance were bacteria seen within the epithelial cells.

Duration of Growth — In none of the cultures did growth occur after seven days. Cilia motion was observed at the end of four days. It is my impression that cilia motion is quickly stopped by bacterial toxins and the accumulation of toxic waste products in the media, and that motion stops long before the cell dies. Mitotic figures were not recognized, however, the amount of growth in some of the cultures exceed the possibility of migration alone.

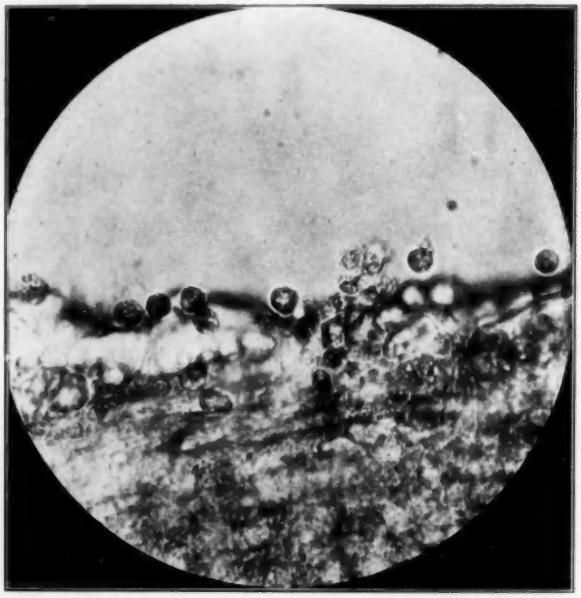

FIG. 1—Tissue culture of Nasal polyp mucous membrane at four hours showing the migration of polymorphonuclear leucocytes from the edge of the explant. High power magnification.

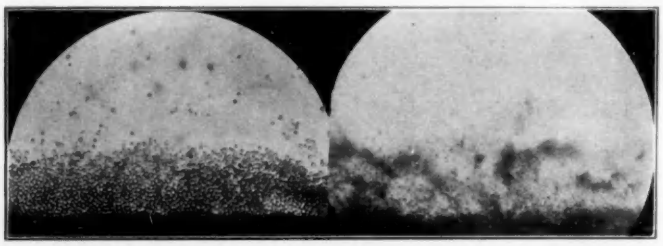

FIG. 2—Tissue culture of Nasal polyp after 24 hours incubation showing the growth of large mononuclear leucocytes and tissue histiocytes at the edge of the explant. Low power.

FIG. 3—Tissue culture of Nasal polyp at 48 hours showing the same field as Fig. 2. There is considerable increase in the amount of growth. L.P.

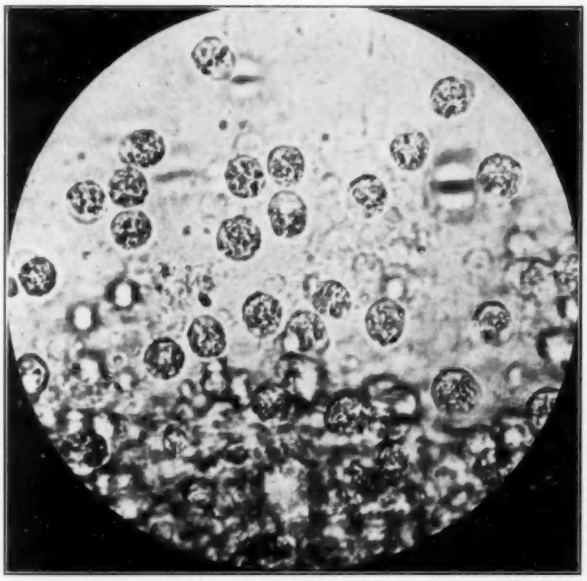

FIG. 4—Tissue culture of Nasal polyp after 24 hours incubation showing large mononuclear cells that have migrated from the edge of the explant. High power magnification.

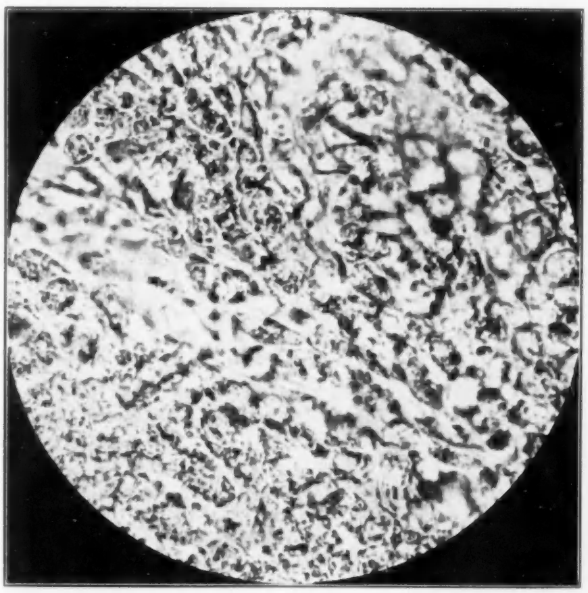

FIG. 5—High power view of tissue culture of Nasal polyp after 48 hours incubation showing the character of growth after the appearance of fibroblasts. The majority of cells in this photograph are large mononuclears and epithelial cells. The fibroblasts are seen as a diagonal streak through the growth and these tend to influence the arrangement of the other cells.

FIG. 6—High power photomicrograph of a tissue culture of Nasal polyp after 72 hours incubation showing cell types at the free edge of the growth. *A*, Large mononuclears or tissue histiocytes. *B*, Desquamated and dead epithelial cells. *C*, Rounded ciliated epithelial cell.

Summary — Tissue cultures offer a valuable means for the study of the behavior and reactions of the various cellular constituents of nasal mucous membrane, and doubtless the solutions of many of the problems relating to the membrane as a whole will be found in the study of these cells in their living state.

Approximately three hundred cultures of nasal mucous membrane were studied, and of the various tissues used nasal polypi gave the most satisfactory results.

In the cultures the cells migrate in the same order as in an inflammatory or traumatic process.

The development of cilia and the formation of goblet cells were not satisfactorily demonstrated. In regard to the latter my observations lead me to feel that the goblet cell is a degenerative process resulting from some chemical alteration of the mucosa, rather than an elective form of the epithelial cell with a secretory function.

In cultures containing bacteria the histiocytes show a marked phagocytosis, whereas the epithelium has a definite resistance to bacteria; these phenomena persisting long after death of the cultures.

My observations lead me to the opinion that the epithelial cell is the first and chief line of defense of the nasal mucous membrane, and that after this defense is destroyed or removed there is little in the secondary defense of the tissues of the stroma that is limited to nasal mucous membrane.

VI. SUMMARY OF SYMPOSIUM

HARRIS P. MOSHER, M.D.

BOSTON, MASS.

THE FIRST PAPER is straight histology and pathology of nasal and sinus mucous membrane. I am proud of the fine slides presented. The paper does not admit of being summarized. It is a paper to be studied by those wishing to acquaint themselves with the fundamental histology and pathology of mucous membrane.

Dr. Schall mentions the observation that in chronically-infected mucous membrane there is often an accumulation of lymphocytes about the glands. Dr. Fenton and Dr. Larsell make the same observation. For the last two or three years, in studying the esophagus, I have been struck with the attraction of the glands for the small round lymphocyte. Often you find a distinct crescent of these cells about a gland or especially about its duct. Only twice have I found active infection of the glands as shown by polymorphonuclear leukocytes in the adjacent tissues.

Dr. Schall states that he feels that cilia are very resistant and quotes me as of the same opinion. I am, since I have found in practically every chronically-infected antrum, no matter how foul, that the mucous membrane shows active cilia. It is usually held that the cilia are easily destroyed. This finding seems to disprove that idea. I have another and even better proof of the resistance of the cilia, which I will give later in connection with the short movie film with which I end my remarks.

The second paper of this symposium has both a scientific and historical interest. In other words, it was this paper which started the discussion, somewhat acrimonious in places, as to whether or not mucous membrane after removal is reformed. The thought back of the paper was to find out whether or not mucous membrane reformed as mucous membrane or was replaced mostly, if not fully, by scar tissue. The slides which you have just seen, however, show the contrary. If you take the

trouble to look up the subject you will find that epithelium makes every endeavor to reform. Physiologists raise their eyebrows when you question the fact that this happens, because they have long known that this is the rule.

I will mention, particularly, but one of the slides; namely, the one with the blood clot extending over the operated cavity as a base for the newly-formed epithelium. This gives the gist of the whole matter. As to whether the newly-formed mucous membrane functions normally or not, nobody of course can tell. However, as it looks normal the supposition is that it acts normally. The new mucous membrane (I am speaking now of antra) comes, of course, from the opening made from the antrum into the nose, or from islands of epithelium left in at the operation. The extent to which mucous membrane is reformed depends upon the amount of residual infection the operation leaves in and about the exenterated sinus.

In the Archives of Laryngology and Rhinology for 1913, Sramoylenko reported the results of animal experiments on the frontal sinus of dogs. He found no trace of reformed mucous membrane. He found further that the frontal sinus, deprived of mucous membrane, became atrophied (partially obliterated) by the formation of new bone, and that this atrophy or obliteration becomes complete in animals within six months or sooner.

Hilding has recently completed some very important work on the reformation of mucous membrane in the frontal sinuses of dogs. His work has been carried farther than the work of any other observer. He has abundantly substantiated the fact that exenterated mucous membrane reforms as apparently normal mucous membrane. Other observers, Bacher and Coates, have also substantiated the fact of the reformation. Hilding, however, has found that the frontal sinus in dogs, after exenteration of the mucous membrane, has a great tendency either to partially or wholly obliterate. He had great difficulty (which bears out the experience of all operators on the human) in keeping the nasal frontal duct open. He made the further observation that the removal of a narrow crescentic strip of mucous membrane taken at right angles to the long axis of the sinus resulted in the formation of a crescentic mucous membrane web. This is the first explanation of these webs, which are so commonly found in the accessory sinuses, which the writer knows of.

Still speaking of mucous membrane, and to complete the important observations of Hilding, the following is mentioned: He closed the vestibule of the nose on one side, using, I think, a dog for the experiment, and left the other vestibule untouched. He records the unexplained phenomena that on the occluded side there was a great increase of goblet cells. On the open side, where the air-blast was too strong, the epithelium changed to the squamous type. The application of this finding is obvious.

The third paper was a follow-up of the second paper. It shows conclusively that after removal of the mucous membrane of the antrum, normal mucous membrane, at least in spots, is reformed. Of course, in removing a specimen of the reformed membrane in the antrum by a punch introduced through the permanent operative opening from the nose into the antrum will give various results. The operator may hit upon practically normal mucous membrane or upon a patch of scar tissue. This is to be expected. The results of any scientific investigation are often prejudiced by the overhead of the laboratory. In connection with the reoperated antra, it has never been found that there was any attempt of the antrum to obliterate. This is a marked contrast to the frequent attempts toward obliteration which Hilding found in the frontal sinuses of dogs.

When investigators reach the point where they can inject into a sinus a given agent and it will be practically always followed by the same change in the cellular elements of the mucous membrane, our knowledge of the function of the mucous membrane will start on the road upward. I think you will agree with me that Fenton and Larsell have gotten off to a flying start in their paper. To date, there has been no experimental histology and pathology of mucous membrane.

All their experiments are noteworthy, three of them especially so. I shall speak of these. It is not as easy as one would think to infect a sinus experimentally. They, however, succeeded in infecting the frontal sinus of a cat with the streptococcus hemolyticus. This was followed by the intradermal injection of a vaccine made from the same organism. In the thickened infected areas the mucous membrane showed an increase of histiocytes under the epithelium. That is, the vaccine, according to the theory of Corbus, marshalled the histiocytes in the mucous membrane at the point of the local lesions. This is an intriguing finding and should be followed up and evaluated.

The double antrum experiment is dramatic. On one side the antrum, as you remember, was washed twice a week for a month with saline solution, and the other antrum at the same intervals, and for the same period had magnesium injected into it. In the magnesium antrum the mucous membrane was very œdematous. Lymphocytes were numerous about the blood vessels. Plasma cells were numerous, also, especially around the deep mucous glands. Numerous eosinophiles were found near the blood vessels and in the tunica propria, where the epithelium was missing. Histiocytes were fairly numerous and there were some fibroblasts. The tall epithelium showed almost every other cell a goblet cell, indicating degeneration. Most of the epithelial cells were still ciliated. Here and there the epithelium was lost and much mucous secretion was present. A few almost normal patches were found.

In the antrum washed with saline the œdema of the mucous membrane was so marked that it appeared to be water-logged. There was marked infiltration of lymphocytes with the formation of patches of lymphoid tissue. The epithelium was degenerated in spots and in places the cilia were absent. The goblet cells were increased. At the places where the epithelium was degenerated there was very little secretion. Lymphocytes were present about the blood vessels. Of the cells present, plasma cells predominated.

In this experiment two different agents were used, and distinct and different findings were found in the mucous membrane. Here then we have a start for further experiment. Here for the first time is something definite in cellular changes to work upon. I cannot help expecting great things from continued work along this line.

There is one immediate application of this experiment which occurs to us all, namely that saline irrigations tend to waterlog mucous membranes. Lille, of the Mayo Clinic, a few years ago presented some experimental work on rabbits along this line, and on his findings advised against the use of saline irrigations or washes. In Fenton's experiment not only was the mucous membrane waterlogged, but the epithelium in places was destroyed.

Fenton's conclusions are here repeated in order to emphasize them. "Subject to further experimental confirmation upon

human subjects, it would seem that the local use of solutions or suspensions of alkaline earth salts or hydroxides (calcium — magnesium) favors the mobilization of histiocytes in acute inflammation. Oily or colloid substances are destructive of epithelium and favor infection by impairing ciliary action, necessitating invasion of the subepithelial stroma by polymorphonuclear cells without any increase in the number of histiocytes. The chlorides seem to favor œdematous changes with marked increase of lymphocytes. An increase of plasma cells seems to accompany long-standing fibrous changes." Fenton has succeeded with his findings in making it harder even than it is at present for the laryngologist to make a living. He discredits both of our old stand-bys — saline irrigations and oil sprays.

Dr. Ross's paper, as you can see by referring to your program, is entitled "A Study of Nasal Mucous Membrane in Hanging-Drop Tissue Culture." In his introduction he states that tissue culture methods have been used in the study of normal and pathological tissue for the past twenty years, but that little or no work along this line has been done on nasal or sinus mucous membrane. Dr. Ross's work on nasal mucous membrane is the first of which I have any knowledge.

He makes the observation that active cilia are found in antral mucous membrane no matter how long or how much the antral mucous membrane is diseased. I have already commented on this observation. He further states that in mucous membrane from the frontal sinus active cilia are not found, and that granulation tissue from the bone is often present. One wonders at once whether this means that the mucous membrane of the frontal sinus is less resistant than the mucous membrane of the antrum, and whether or not the frequent occurrence of granulation tissue in the mucous membrane of the frontal sinus may not give a hint as to the cause of the difference in frequency of osteomyelitis of the frontal sinus, and osteomyelitis of the superior maxilla.

Dr. Ross developed one new point in connection with the technique of making tissue cultures. Namely, in place of using an embryonic extract he uses fresh umbilical cord, and this seems to work well. He did not succeed in keeping the cultures alive beyond seven days. The longest time active cilia were observed was four days. He feels that they are stopped by toxins

and cease moving before the death of the cell. Dr. Ross found that the order of the migration of the cells is the same as it is in inflammation and after trauma. It would seem that he was almost on the point of discovering how cilia are formed. Up to now this has not been explained.

His most interesting observation concerns the epithelial cells. These he found are not readily attacked by bacteria, whereas the histiocytes, whose business it is to attack bacteria, do so at once and then soon die.

Dr. Ross apparently is of the opinion that goblet cells are the result of degeneration. Larsell seems to be of the same opinion. This, however, is not the orthodox belief.

The concluding statement, that the epithelial cells offer the first line of defense, is somewhat at variance with the opinion of Fenton and Larsell, who maintain that this honor should go to the histiocytes.

Dr. Ross's final statement that the study of tissue cultures of sinus mucous membrane offers the solution of many of the problems, physical, chemical, and biological, which at present confront us in our study of disease of the sinus mucous membrane, I heartily agree with.

In studying mucous membrane, especially in watching for cellular changes, it is well to watch the area just beneath the basement membrane. This is the main objective of the blood supply. The vessels enter the substance of the mucous membrane from the outside at rather regular intervals, and take a straight course toward the epithelium. They arborize below the basement membrane and then run both ways, paralleling the membrane. Here then is the center of the blood supply. This probably explains the frequent occurrence of a strip of small round lymphocytes below the basement membrane in chronic infection. It is at this point that solitary follicles occur in the esophagus and intestines. The mucous membrane of a chronically-infected tear sac shows these changes very nicely, namely, that there is an intense infiltration of small round lymphocytes with an occasional lymph nodule directly under the epithelium and basement membrane.

In this symposium the papers have dealt chiefly with the reformation of mucous membrane, with the growth of mucous membranes in cultures, and with cellular changes produced by

experimentation. Also there was the groundwork paper on histology and pathology. But little has been said about the glands. I have long had the feeling that the toxins elaborated by mucous membrane, if any toxins are produced by it, are due to an altered chemistry of the glands. At the moment this subject is another darkest Africa awaiting its Livingston.

One other point in regard to mucous membrane, and I am done. Larsell and Ross, Ross especially, have the feeling that goblet cells are a form of cell degeneration. I should like to see this fully proved.

THE PLACE OF DIATHERMY FOR TONSILLECTOMIES

SAMUEL R. SKILLERN, JR., M.D.

PHILADELPHIA, PA.

SINCE THE pioneer work of Clark, McFee, Doly and others, surgical diathermy has advanced by leaps and bounds, both in the production of this type of current and in its application in general, as well as in the specialized branches of surgery; but the concensus of opinion seems to be that this manner of destroying living tissue will never supplant the recognized surgery of our predecessors.

Portmann and Moriau[1] of Bordeaux, France, in their paper of 1924, were among the first laryngologists to put the stamp of approval on the removal of the tonsils by diathermy. Four years later Frecker[2] of Australia wrote on the ease of this operation, but I am afraid I[3] shall have to plead guilty to the fallacy of starting the deluge of articles that are flooding our literature.

When my paper was presented before the Virginia Medical Society in the spring of 1928, electrocoagulation was not advocated as the operation of choice, rather an operation of necessity in the otherwise inoperable case.

Inquiring among those laryngologists who have added electrocoagulation to their equipment, it seems they are using it exclusively for the removal of tonsillar stumps or tabs of lymphoid tissue that so often recur in the throat of the tonsillectomized patient, and for the shrinkage of hypertrophied lingual tonsils, but orthodox surgery is their preference for a complete tonsillectomy.

In Philadelphia we are receiving numerous inquiries from our patients about this so-called bloodless, painless operation by which tonsils can be removed without anesthesia or without requiring them to take time from their daily occupation. What has brought about this condition? Simply the extensive advertising campaign by some of the manufacturers of the high frequency cabinet.

I have with me circulars that were received by several of my patients from a Philadelphia agency expounding upon the safety and economy of removing the tonsils by electrocoagulation. Consequently, during these times of depression the average patient cannot be censured for seeking the operation of least expense, and, in this ignorance, of selecting this procedure in preference to the orthodox operation with its loss of time and hospital expenditure.

For the physician, this company has a regular follow-up system of cards which infer that tonsillectomy by electrocoagulation is such a simple, easy procedure that anyone can readily master it by attending a few of their clinics. The consequence is that nearly every rural, and a great many city physicians, who would not otherwise attempt a tonsillectomy, are trying to remove tonsils by coagulation. I have heard a specialist defined as "one who knows more and more about less and less," and I feel sure that as these well-meaning but disillusioned general practitioners learn more about tonsils and their removal, there will be less of these electrocoagulating machines sold.

Beals,[4] in his article, stated: "The general practitioner is misled because he believes he can easily master this method, and the unethical manufacturer adds to the chaos by sending out high-pressure salesmen who would sell any prospect regardless of the operator's qualifications." Coates,[5] in discussing this paper, upholds the thought as advocated in my original paper; i.e., "Every laryngologist should include electrocoagulation in his operative technique to keep this method out of the hands of the charlatans and those physicians who consider only the monetary gain. Failure of the trained tonsil operators to include this technique will simply drive our patients to such men, as many members of the general profession are cognizant of this method and are advising it for patients whom they considered as inoperable."

In 1927, I[3] stated that electrocoagulation required as much skill and knowledge of the parts as any of the other surgical procedures.

Dillinger[6] states that "This method requires more skill and training than the ordinary surgical tonsillectomy, and no one except a throat specialist should attempt to use diathermy in the throat."

After five years of usage, in the selected type of case, I fully agree with him, as to me it is the most difficult and exhausting method of removing the tonsils. The first few treatments, especially of the bulging type of tonsil, are as simple as the coagulation of the raw beef (the usual salesman's demonstration), but after the majority of the tonsillar tissue is removed, each subsequent treatment is more difficult than its predecessor. To remove the last vestige of tonsillar tissue buried behind a stiffened anterior pillar, with the patient gagging, thus burying the needle deeper than desired, requires all of my skill and judgment to prevent injury to the musclature or to a possible pulsating vessel in the base of the fossa.

The postoperative results depend entirely upon the skill and conscientiousness of the operator. Just as complete a removal, and as satisfactory a postoperative result can be obtained by the careful operator as in any other surgical procedure, but as Myers[7] says, "It is no method for the neophyte."

Whoever says that the removal of the tonsils by electrocoagulation is a painless procedure is, in my opinion, twisting the truth to fit his own desired belief. No matter what part of the anatomy is destroyed, or how, it will resent the insult as long as the conscious nervous system is functioning.

The pain is never of the unbearable type, but there is postoperative pain and what becomes often even worse is the apprehension of the following treatment, which may become an actual dread.

Allow me to cite you several authentic cases of the experiment of removing one tonsil and coagulating the other and of tonsillectomy after a partial coagulation.

Two of these cases were anesthetized by novocaine injection. The right tonsil in one patient, the left in the other were removed by snare and dissection. The remaining tonsil was coagulated. In two other cases one tonsil was injected and removed by the LaForce instrument, and the other tonsil coagulated. All four of these cases stated the greatest postoperative pain was in the coagulated tonsil.

In six cases that had had from one to five coagulations, the tonsils were removed by LaForce. In all but one instance the patients stated the immediate pain was no greater after total extirpation than after each coagulation, but the soreness was of

longer duration. I have slides showing the amount of tissue destroyed by coagulation in each tonsil.

I agree fully with Coates[5] that electrocoagulation is not the operation of choice, but in the so-called poor operative risk, when the fear of operation or the inability to take sufficient time from work for hospitalization is a factor, electrocoagulation is the operation of necessity.

That this procedure is not without operative dangers and postoperative complications is evidenced by the reporting of thirty casualties in those interviewed by Myers.[7]

The placing of the necessary equipment in the offices of the general practitioner, or any one who is not familiar with the surgical use of electricity, is little short of malpractice, as no one should attempt the removal of the tonsils by electrocoagulation unless he is capable of removing them by the recognized surgical techniques and meeting any emergency or complication that may arise.

It is my opinion that the potential dangers of electrocoagulation increase in multiple in proportion to the lack of knowledge and the lack of familiarity of the surgical anatomy of the throat, the minute anatomy and pathology of the tonsillar tissue, and the complications and sequelæ that may occur.

CONCLUSIONS

1. The laryngologist should familiarize himself with the technique of electrocoagulation for the selective type of case.
2. Electrocoagulation should be considered as an operation of necessity and not of choice.
3. In the hands of the inexperienced it is the most difficult and dangerous method of removing the tonsils.
4. It should be handled with great caution, with strict limitations of the current, and only by the experienced expert hands of the laryngologist.
5. The general practitioners should be warned of the dangers of electrocoagulation, and also of the high-pressure salesman's racket.

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DISCUSSION

DR. ROBERT F. RIDPATH, Philadelphia: Dr. Skillern has, in my opinion, developed three very important facts. First, that the electrical destruction of tissue will never supplant the recognized surgery of our forefathers; second, the tremendous propaganda which the manufacturers of various electrical apparatus are using through their agents to frighten the laity and doctors, in a sense criticizing the use of surgery for tonsillular removal; and third, through this psychology compelling the physician to augment by their armamentarium his already augmented paraphernalia.

That there may be cases where electric coagulation is preferable to the surgery generally employed, I personally doubt. In my many years of experience, including work in three large hospitals and dispensaries, I have never had a case where indications warranted tonsillar removal that could not be done by a specified surgical technique. My cases have run the gauntlet of age and disease — from a child two years old to a person eighty-two years old, including acute nephritis, diabetes, tuberculosis, heart lesions, hemophilia, bed-ridden patients, etc.; from those in decidedly precarious condition to those who were ambulatory, and although admitting that there may be certain conditions found in isolated cases which justify the destruction of the tissue by electric processes, I, personally, have never met such a case.

The second phase of Dr. Skillern's paper deals with the suggestion that it were better for the qualified physician to add electrocoagulation to his armamentarium than to permit its use by others than the recognized laryngologists. That, I feel, is a point well taken. That the manufacturers of the various electric dessicators for electrocoagulation have a well thought out system for advertisement, not only to the physician, but to the

laity in general, is a fact all too apparent in many cities, of which Philadelphia is one. I shall read from a few circulars which I have with me:

"Dr. Charles J. Muttart and assistants, 1813 Pine Street, Philadelphia, announce the opening of a clinic, 3816 Locust Street, specializing in non-surgical methods of removing diseased tonsils, rectal diseases and women's diseases. No charge for examination. New patients received on Tuesdays as follows: Rectal diseases, 2 to 3; tonsil cases, 3 to 5; women's diseases, 7 to 8.

"The non-surgical removal of these diseases has been enthusiastically accepted by physicians and laymen who have seen the results. It is no longer necessary to spend time in a hospital or undergo anesthesia.

"The treatment method is simple, harmless, painless and bloodless. Patients carry on their respective vocations without losing time from work, business or social activities.

"As you know, diseased tonsils are responsible for many cases of rheumatism, heart disease, nephritis, deafness, etc.

"Fees for treatment are moderate and are fully agreed upon before treatment begins. Treatments are given at weekly intervals.

"We trust that you will not hesitate to avail yourself of this opportunity to be examined without charge."

"Doctor: Don't add your name to the long list of physicians who are at this moment 'loaded up' with high voltage machines of antiquated engineering design, and who have not been successful in finishing up tonsil cases.

"Good end-results in coagulation require proper technic obtained from physicians skilled in the work, and who can show you finished cases. Would it not be wise to see patients in various stages of treatment? See competent, ethical men coagulating in rectal, cervical, and throat work. See the little kinks that make for the desired end-results and satisfied patients.

"Every American Coagulator is in use: Not dusty from non-use, because each physician attended clinic under the supervision of experienced medical men.

"Why not start right? See the work. Talk to the patients. You will never regret taking up the work!

"Register now for the next clinic. Fill out and mail the attached card, and dates will be sent to you. American Medical Apparatus Co., 3816 Locust Street, Philadelphia, Pa. Near the University of Pennsylvania. Unlimited parking."

"Doctor: The intelligent layman now exercises his right to choose between two methods of Tonsillectomy, Surgery or Electrocoagulation. In order to avoid surgery, the patient is willing to pay a larger fee for the less hazardous method. (Medical substantiation of this fact is available.)

"Those physicians who utilize Electrocoagulation are not depressed with empty reception rooms.

"Professional opinion has swung to a non-surgical method, as is

evidenced by this technique being added to the curriculum of a great school of Medicine in Philadelphia.

"Why lose your patients to offices that are equipped — why limit yourself to small fees and permit the operating fees to go elsewhere?

"Electrocoagulation is successful in cervical, rectal and malignancy work. The complete technique can be obtained through a clinic under competent, ethical physicians, without charge.

"You are invited to register, now. American Medical Apparatus Co., 3816 Locust Street, Philadelphia, Pa."

I will ask Dr. Skillern, in closing the discussion, to read a letter which he has.

They, the agents of these machines, are most persistent in their talk and insistent upon your purchase of their particular electric apparatus, and do not hesitate to use the terms: "old fashioned," "out of date," "behind the times," and "you will most surely regret your stand," in their propaganda talk.

Although not doing or advocating electrocoagulation for the destruction of tonsillar tissue, I can imagine, although I am using my cerebration to the breaking point, that there may be certain cases when this procedure is justifiable when done under the scientifically matured hands of an experienced laryngologist.

My first experience of a case who had had his tonsillar tissue electrocoagulated happened to be a friend of mine, a lawyer of repute, who, although knowing my specialty, received the idea of electrocoagulation from another friend of his and decided to undergo the process. Several months afterwards he consulted me as to his continuous painfully inflamed throat. After making a careful examination, I suggested a tonsillectomy, when he replied, "Why, I had my tonsils removed by electricity." There was, unfortunately, nothing else for me to do except to tell him the truth — that although he may have had electrocoagulation of his tonsils, nevertheless the tonsils were still present and I advised their removal under surgical means. This was done, and I need hardly state that the operation was a very difficult one on account of the increase of fibrous tissue from the partial destruction by electrocoagulation, and the recovery was extremely slow because the trauma caused by a necessarily most thorough dissection.

This case is typical of many which have since come under my care. Let me quote you another case of a well known man, a member of this Academy, who called me on the phone one Sunday morning about eight o'clock, asking me to come to my office to see him. Upon arrival I found the patient had in the past three weeks had three treatments for the removal of diseased tonsils by the electrocoagulation method by another member of this Academy. The patient was almost speechless from loss of blood and in a precarious condition, and although having all the necessary instruments at my disposal, it was some time before I was able to stop the flow of blood and only then by the removal of the offending tonsil by surgical means.

I therefore conclude that, allowing there may be cases where electrocoagulation is the method of necessity (in cases who for some reason are considered inoperable), I personally have never met them.

DR. JAMES A. FLYNN, Washington, D. C.: In regard to this method, I want to put Professor Portmann in correct position before the Academy. He introduced the method, but not to supplant surgery. He recommends it only in cases where surgery is contraindicated. During the considerable time I have spent in the Bordeaux Clinic in the past six years I have never seen it used. Professor Portmann personally stated to me that he would consider it a dangerous method for routine use.

There is an insidious propaganda being circulated in newspaper reading-notices to the effect that tonsil surgery is dangerous; that the patient assumes the danger of hospitalization, of hemorrhage, lung abscess, etc., and then suggests that the patient find some method of treatment other than surgery. An address is mentioned in the newspaper article, generally a postoffice box number. On writing to the address given one receives a circular in return instructing him to communicate with a particular doctor or chiropractor who possesses a machine made by this particular company. Recently I saw such a circular. It was brought to me by a patient who wanted to know if I thought the method was good. It had been highly recommended by a chiropractor, who had the only machine in Washington made by this company. Such methods of publicity are being broadcast throughout our country, attempting to create an unfavorable impression concerning tonsil surgery.

I wish to emphasize the point that Professor Portmann does not recommend diathermy as the method of choice for tonsil removal, but advocates it as a method for a very limited number of cases unsuitable for surgery.

DR. GEORGE L. RICHARDS, South Yarmouth, Mass.: One thing Dr. Skillern omitted. The last speaker mentioned newspaper articles. I recently had as a patient a newspaper man who suggested that his tonsils be taken out by electrocoagulation. I said, "Go ahead and have it done. I should like to see how your throat looks afterwards." I then suggested that he look up cases reported to him as having succeeded. He did so and found that about half of them never finished the treatment; they tired out and gave it up, inasmuch as it seemed they would have to continue treatment for a long time. So this man decided he would have his done by a method that would not take quite so long, and while it might lay him up for a few days it would be over with. His particular case was finished quickly and satisfactorily and he is all right now; and he is no advocate of electrocoagulation.

DR. E. M. JOSEPHSON, New York: My reason for entering the discussion is that I do not think Dr. Skillern and Dr. Ridpath have sufficiently emphasized the falsity of the claim made by the manufacturers of diathermy machines that there are no dangers attached to electrocoagulation of the tonsils, and in particular that there is no danger of hemorrhage. I first tried electrocoagulation of the tonsils in a series of cases in 1925.

The cases in the series repeatedly developed delayed hemorrhages ensuing days after the treatment. I recall one case in particular in which it was found to be impossible to stop the oozing hemorrhage which started when the scab of the treatment came away, until the patient was almost exsanguinated. I was interested to note that Dr. Lee Hurd of New York recently reported his experience with electrocoagulation, which accorded with mine.

Another disagreeable by-effect of electrocoagulation which the exploiters belie is the pain frequently consequent upon the treatment. In infected tonsils it is not at all unusual for the patient to develop pain as acute as that of a peritonsillar abscess following each electrocoagulation treatment, which incapacitates for days.

As an antidote to the unscrupulous exploitation of electrocoagulation it seems to be wiser to tell the public the whole truth of the matter rather than launch an attack which might appear to be directed at confreres and to have the semblance of a selfish attempt to undermine them.

Dr. Samuel R. Skillern, closing: My object in presenting this subject to the Academy was to put electrocoagulation in its proper place. It has its place in otolaryngology, as it has in general surgery, as a very useful adjunct to our operative procedures. I still feel that the laryngologist should include electrocoagulation, as it *is* a method of removing the tonsils, and in the skillful hands of the conscientious operator a complete and thorough enucleation of all tonsillar tissue can be performed.

Personally, I do not favor electrocoagulation, and although I have removed a number of tonsils by this method, I never recommend it as the operation of choice.

All of my electrocoagulation cases have been referred by colleagues with the request that this patient's tonsils be removed by this method, or else the patient has insisted upon this procedure; but I always try to persuade patients to accept a LaForce operation, telling them that I would not have my tonsils removed by this fractional method. Before the complete removal was accomplished (by electrocoagulation) the patients told me they were sorry they had not accepted my advice, and if they had it to do over again they would have their tonsils entirely removed at one operation.

Each week as one coagulates one tonsil there is more or less suffering, and when you consider that it may require from ten to twenty weeks to complete the operation you can judge for yourself what the patient has to go through.

I am very glad to hear that Dr. Portmann is no longer advocating this method as the operation of choice.

Dr. Ridpath has asked me to read you a letter that many of my patients have received.

"Dear Madam:

"If the sufferer knew as much as the doctor about the five major risks attending the removal of tonsils by surgery, there would be fewer operations by this method. As it is there are fewer every day.

"The risk of death over etherization or injection, of hemorrhage and heart complications, and most insidious lung abscess due to infection getting into the lung during the operation or immediately afterward, is recognized by every surgeon.

"According to Dr. Chevalier Jackson, this lung abscess is the result of the tonsil operation performed surgically sometimes months before.

"This company produces the apparatus required for the bloodless, painless removal of the tonsils without ether, knife or gas, and without loss of time from your work or activities.

"The machine is used in hospitals and in the offices of nationally known specialists.

"It never pays to delay. Why not permit us to refer you to a doctor skilled in this procedure? He will make no charge for a throat examination and advice.

"If you know anyone who is unable to pay the regular fee for tonsil removal, this company has arranged with a nonsurgical clinic to treat such patients at clinical prices. Physicians of the greatest skill devote their time to this clinic.

"Patients may call at 3816 Locust Street, side clinic entrance, and consult with the physician without charge between the hours 10 a.m. to 1 p.m. on Thursdays.

"Cordially,"

I wish to go on record, Mr. Chairman, not as an advocate of this method, but as one who recognizes its advantages as a method of removing the tonsils in a selected type of case that comes to every laryngologist for tonsillectomy.

TEACHERS' SECTION

INSTRUCTIONAL SUBJECTS

PRESENTATION OF INSTRUMENTS

MINUTES

NECROLOGY

MODERN TREND OF UNDERGRADUATE MEDICAL TEACHING

DR. C. F. MARTIN, *Dean, McGill University*

MONTREAL, CANADA

MR. PRESIDENT AND GENTLEMEN: I need not begin with the usual word of gratitude, although I feel it very much. It is an honor to meet with you tonight, and it is a particularly pleasure to know that you are meeting in the university center where years ago Dr. Frank Buller occupied the chair of ophthalmology. He was in his own specialty at that time greater than any other man in any other specialty in this country. His memory lingers with us and will for many a year.

In otolaryngology, under the guidance of Dr. Birkett for many years, our department obtained a prestige through the international character of his work and his reputation. We have every reason to believe that those who are succeeding these two great men, Drs. Byers and McKee, and Tooke and White, will follow in the steps of their predecessors and that these departments will retain the prestige secured under the régime of Drs. Buller and Birkett.

As I looked at the program I thought I was asked to speak on "The Trend of Undergraduate Medical Teaching," and I took it to imply the general education of the undergraduate; but I find on coming here that perhaps I was mistaken and that I am supposed to speak more concerning undergraduate teaching in relation to ophthalmology and otolaryngology. I shall, however, leave that for the last item of my remarks, and your president, Dr. McKee, says I am leaving the best for the last! Nevertheless, in approaching this subject I am reminded of the story of the lady in a motor car (of course, it was a Ford) whose vehicle was jammed in the traffic on Fifth Avenue, in the busy hour of the day. The officer halted her, first, for running past a light, and second, for having her car pointed in the wrong direction. The policeman came up to her and said,

"Lady, do you know anything about the traffic regulations here?" "Oh, yes, a little," she replied. "Can I help you in any way?" My own feelings are very much akin to those of the lady. I do not know that I can help you very much, but I would like to say in general that I believe we in the medical schools agree that whatever the trend may be, it is for the better. Everywhere physicians are being better trained, better educated, better equipped; disease is better understood, it is more thoroughly studied and better treated, and diminution in human suffering is indeed such as to call for the approbation of the angels. On the other hand, do not let us forget that the autocracy of the cults, the public's faith in miracles, the constant desire of the people to try irregular methods of practice, is perhaps increasing, and that, too, in face of the wonderful achievements that regular medicine has made in the last decade.

I take it there are three evils that hamper the progress of medical education, and these refer in the first place to the schools; second, to the teachers, and third, to the students.

Medical schools, it is universally agreed, are everywhere straining to avoid an overcrowding of the timetable, and at the same time are adding more and more sub-departmental subjects to the curriculum. As, for example, the appointment in some universities of professors of proctology, of dietetics, and the like. Teachers, on the other hand, in their eagerness to give everything they can, that no subject may suffer from neglect, are constantly demanding more and more hours for teaching. That applies to every department and the poor deans have to suffer accordingly. I am reminded of the quotation from Goethe: "Who never ate his bread in sorrow will not know what suffering is."

So far as the *students* are concerned, the subjects are now so vast and complicated that I feel sometimes the students are doing their utmost to "get away with" as little as possible in the way of information in order merely to satisfy the requirements for examination. That comes, I believe, from the fact that the subjects are so many and complex and the teachers are trying so hard to teach everything they can, and are consequently crowding the curriculum to an impossible extent. It is thus impossible for the student to get anything but the minimum of learning, the minimum of information that will secure for him

his final degree. This is not "education!" Fortunately, in every school the ideals are the same. There is no doubt of that. It is the method that differs. In every school men differ in their power to impart and stimulate, it matters not in what school or what place. They are always open to criticism that is adverse, and in many instances it is not warranted. But as I have already indicated, the physiological divisions of the sub-departmental subjects have become pathological; in some departments there is necrosis and degeneration; in others there is hypertrophy and outgrowth — both a menace to medical education. Standards have been raised in every school, and it is in the effort to maintain the standards that we are all going this pace and allowing, in our curricula, minor departments an independence that menaces the facilities for giving a good medical education. Perhaps we are losing our perspective — I am speaking of schools in general. We are apt to do this — we are apt to make additions to our timetable and curriculum without making selection. If we were to select as we go along, cut out the unnecessary, give the necessary, it would facilitate teaching and give teachers and students an opportunity to carry out a more ideal program.

May I say here that we, here in McGill, have prided ourselves on being a great clinical school. Since 1821-22, when the school was founded with a charter that demanded bedside teaching, that tradition has been carried on, so that while laboratories have their places in the development of medical science, the clinical aspects have been the ones in which we have been mainly interested from the point of view of undergraduate medical training. Osler learned that method at McGill and carried it with conspicuous success, as you are aware, to the various universities of the United States.

We believe the student should be responsible to a large extent for his own education; that he should extract the knowledge as best he can without being too much led by the hand or nursed along as they do in the case of vocational training to the mentally or physically handicapped. We do not believe this is the education that will make scientific practitioners of our students.

What are the medical schools in this continent interested in? They are first of all interested in the improvement of secondary schools. We would like to see the schools taking on a little bit

more than they are, that these boys coming to the university shall have had more of the things which are now given in the first year of arts. That would tend to shorten the curriculum hours of work and help by giving a more cultural program in the arts classes to minimize the amount of science demanded in pre-medical work. You who are ophthalmologists are particularly interested in early training in physics. But how little does the man who comes into ophthalmology remember of the physics he learned in his boyhood? Very little. He has to learn that again, but it will be easy if he has had the elementary principles in the grades. How much electricity does he remember and apply to the electrocardiograph? Not very much. We believe in McGill that when a boy comes up to medicine he should know something of the classics; otherwise, the study of anatomy becomes a feat of memory. And if he does not understand the derivation of words he makes a poor fist of rational anatomical training. And that is very important.

So far as the preclinical subjects are concerned — anatomy, physiology and biochemistry — there is a very old controversy as to how much instruction the students should have. It was our impression in the early days that anatomy could not be mastered with less than two years of close study. That was my experience as a student, particularly under Drs. Shepherd and Birkett, and we learned a great deal of anatomy, it is true; but in those days many sciences were absent from the curriculum that are present today. We had no biochemistry, very little hygiene, very little laboratory work. So we believe, as do many of the universities in the United States, that it is a good thing perhaps to make the study of anatomy somewhat more elementary, but make it as good a training as is possible within the time — teach them to make careful dissection of a limited number of parts of the body. We feel that anatomy should not occupy quite as large a part of the curriculum as has been the custom in Great Britain. We do not believe the time devoted to it is commensurate with its importance. For that reason we have cut down the study of anatomy to one year of general training, and in the second year the students have an option of continuing the work three afternoons a week throughout the session.

Universities are questioning the importance of detailed study of biochemistry and they are beginning to wonder if perhaps

physiology and biochemistry might not be given to the undergraduates under one combined course of study. Physiologists should know enough of biochemistry to give training in that subject to undergraduates. If the two are to be divorced, would it not be wiser to make the study of biochemistry rather elementary so as not to take up too much time and yet give the student enough knowledge for his clinical years where he is given further instruction in this subject. Dr. Pepper spoke this morning along this line and he is one of the best advocates of that idea. He has done some wonderful work in Philadelphia in associating the preclinical sciences and clinical medicine in a way that is most instructive to the men, and doubtless very instructive to the teacher, too.

There is another point about which controversy arises, and that is how early patients should be admitted to the observation of students. There are those who admit them through the first year, as they do in France. In the French hospitals the first thing a student does is to go to the wards and take histories of cases observed. That has not been our custom, but we are beginning to think that perhaps it is wiser to have the patients exposed to the students, and vice versa, earlier than the third year; that they might take more human interest in the patient, for that, after all, is what we seek. When the students come to the university, we will say McGill, many of them have the idea that they come for the purpose of ending for all time the ills of humanity! They have a thoroughly human interest in the practice of medicine, but soon they find there is a steep and thorny road before they get to study the patient, a road that leads through the dissecting room, through physiology and biochemistry, and by the time they come to the clinical years they have lost much of their zest for the human side of medicine, since they must depend on the laboratories to such a large extent for the diagnosis of the patient. So often when the attending physician enters the ward for the purpose of examining a new patient his attention is all too readily drawn to laboratory examinations or blood chemistry, or a few other things of that kind, deferring the diagnosis and treatment indefinitely. The old routine clinical examination, where the human patient is involved, is apt sometimes to take on secondary importance because the laboratories have a way of dominating so that the clinician depends too much on that

source of observation. No clinician, of course, will admit such a criterion.

A word about the specialties in the undergraduate curriculum, and about specialties in general. This does not include ophthalmology and otolaryngology! The public demand for specialists may be quite natural in theory, but not in practice. The habit is increasing to demand a specialist for each organ and a physician for each disease. I have no quarrel with the expert whose daily routine is confined to the extraction of the unerupted second molar teeth; his technical skill justifies his effort and his large income. Where skill and craftsmanship is concerned, give him the undisputed field; but he must not monopolize the whole field of medical knowledge. The patient is not simply a collection of organs; there is the man, the woman, or the child in the issue. Divorced from complete clinical examination, specialties are bad in principle and dangerous in practice. Hence the trend today in medical schools to emphasize more and more the three major subjects — medicine, surgery and obstetrics; to make the specialties subordinate. I refer to orthopedics, urology, neurology, dermatology, and various others which should be made more subsidiary. I do not refer to ophthalmology and otolaryngology. Specialties such as orthopedics and others I have mentioned should be taught by the general surgeon, and by the general physician himself to the undergraduates, as a part of general medicine and surgery. That should be the tendency today in medical schools; it is the tendency expressed recently by the Council on Medical Education of the American Association. But the opportunity should exist in every school, in every hospital, for more detailed knowledge in these subjects, and the course should be made optional or for postgraduate study; in that way only do we believe there will be a proper appreciation of the value of various subjects in the training of the undergraduate.

I have not come prepared to say more than a word about ophthalmology and otolaryngology, and that word is this: that we believe in McGill, as in most other schools, that these subjects of course require an adequate number of demonstrations, lectures, etc. As to the time to be devoted to these subjects, it is conceded by many that the technical study involved in the technique of examinations should be introduced early in the curric-

ulum, in, if possible, the earliest part of the clinical work. We also believe that the education should require a minimum amount of time, sufficient to give the undergraduate that knowledge of the eye and ear which the general practitioner will in a general way find useful. But it is utterly absurd, of course, to expect such knowledge as will make of such a man a specialist, and yet there are indications that too much is required. With such views we take issue. I do not think any one of you would be willing to place in the hands of such a man the responsibility for things of that kind. But if the student is taught the elementary things for general practice, as much as possible by demonstration, as Dr. Byers and Dr. McKee and Dr. White and his assistants are doing — as much as possible at the bedside, he will obtain the maximum of practice and the minimum of theory. Whether the amount of demonstration and lectures should be twenty or sixty hours, I do not know, but I do not think you can standardize education in that way. Some men with certain opportunities could give all the instruction that would be required in twenty hours; others would not do it in one hundred. You have to study the need, the teacher, and many other things. And if anybody tells the dean and the faculty he cannot do it in less than sixty hours I think you have to consider whether conditions demand that in the school. The same thing applies to otolaryngology.

One thing is certain, that in general medicine and general surgery there should be every attention paid in the surgical and medical wards to a consideration of the eye and ear. Either the specialists should bring the students into the general wards, or the patient should be brought to their clinics. But the relation between general medicine and general surgery and the eye and ear cannot be stressed too much, and I think will have a tremendous effect on the general practitioner.

I thank you for your kindness in asking me to come here and for your patience in listening.

SURVEY OF UNDERGRADUATE OPHTHALMOLOGY

HARRY S. GRADLE, M.D.

CHICAGO, ILL.

When the Teachers' Section was started several years ago there were no definite ideas as to what could be accomplished and what could be discussed in such a Section. It has taken some time for the Section gradually to sift out the desired ideas and evolve a general scheme of work. This year the idea is to confine our discussion to undergraduate teaching of ophthalmology and otolaryngology. To that end there were two committees appointed to investigate, as far as possible, undergraduate teaching in the two specialties, analyze the teaching in the various institutions, and see what recommendations could be evolved. A questionnaire was sent to sixty Class A medical schools, and undoubtedly to most of you, in which we endeavored to find the numerical strength of the faculty, the number of hours devoted to the subject, the amount of didactic work and the amount of clinical work, the subjects presented, etc.; in short, a general survey of the teaching. From these answers an analysis was made which you find on the mimeographed sheet before you. I do not think there is any necessity to go into any lengthy discussion because it is self-evident, but I may speak of the results of the analysis of these replies and the recommendations to be made.

In the first place, it is rather our impression that too many of the departments of ophthalmology in the medical schools are run for the instruction and possibly the aggrandizement of the chief of staff of the eye department and his assistants; that in some instances more attention is paid to the possibilities of instruction of the instructor than of the students; that not sufficient time is devoted to the clinical and personal instruction of the students; and that the endeavor is made in too many instances to show students the unusual rather than the ordinary, everyday occurrences that they will meet after they leave the halls of the institution. Of course we must say that many of the answers were idealistic rather than on a purely material basis, and some had to be taken from a first-hand knowledge of the school.

From the replies to the questionnaire, and from the committee's experience in teaching, we have taken the liberty of making the following recommendations as a rather broad basis for the undergraduate teaching of ophthalmology, recognizing of course that undergraduate teaching of ophthalmology be with the basic idea that the student after leaving the college and the hospital is going into general practice: Such undergraduate student must be given the instruction in ophthalmology that he may need for general practice, and any specialization is entirely out of the

question until the general education of the student has been rounded out. We do not believe the undergraduate student is capable of selecting the subjects he needs for his future life; we believe that his course should to a great extent be laid out for him. He does not know how much time he ought to give to ophthalmology, to gastroenterology, to orthopedics, or to neurology, and he must accept the experience of the deans in determining what is best for each man. But we do believe that ophthalmology should be a compulsory subject, both in didactic and clinical phases, and we do believe that ophthalmology should be a somewhat isolated department, neither subsidiary nor subservient to any other, but one of the independent departments of the medical school. We do not intend to say that ophthalmology should occupy a position on a par with medicine, surgery and obstetrics, but we do believe it should be regarded as a separate department with its own staff, and a separate budget for such maintenance as may be needed. We further believe the department of ophthalmology should cooperate with the departments of anatomy and physiology for the first two non-clinical years. In other words, a certain amount of time should be given by the departments of anatomy and physiology to the teaching of this subject, with the active cooperation of the department of ophthalmology. We believe the ideal undergraduate course should embrace sixty hours of work, as accepted by the Association of Medical Colleges; but we realize that is rather a broad basis and in the majority of schools sixty hours is impossible. Consequently, recognizing that that amount of time is not feasible, we are making our suggestions for the proposed broad ophthalmologic course upon a lesser number of hours, realizing the difficulties that the dean has in coordinating the hours for small sections, and coordinating the hours of the faculty. Therefore we recommend that fifty hours be the minimum, divided as follows: Ophthalmology, two hours given at the very beginning of the Junior year; in the first clinical year the student should use the ophthalmoscope in order that during his clinical years he may use the ophthalmoscope diagnostically as he uses the stethoscope — a matter of routine. The use of the ophthalmoscope should be taught in small sections. We further believe that thirteen hours should be given to didactic ophthalmology, instructing the student in the necessary principles of ophthalmology, divided as you see it here. The remainder of the time should be given in the third or fourth year.

Clinical ophthalmology should be given in outpatient work; it should be given in small sections, and each section should be assigned to an instructor relieved from other duties so that his sole duty will be the teaching of the students in that section. These students should be instructed in methods of examination of the patient; and they should be instructed in the commoner diseases of the eye as they may meet them in their future life as general practitioners. They should be instructed what cases they should handle and what would be dangerous for them to handle. We do not believe the student should go beyond that in undergraduate ophthalmology. If time permits, the student should avail himself of two elective courses — advanced ophthalmoscopy and advanced clinical ophthalmology. We do not believe that in undergraduate life research in ophthalmology

should be encouraged among students. We do believe, of course, that the department of ophthalmology should employ the modern visual aids to teaching, motion pictures, lantern slides, etc.

We recommend that no instructor be accepted in class A medical schools without the certificate of the American Board for Ophthalmic Examinations.

The committee offers this report to you for discussion, in the hope that in case you decide to accept any or all of the report and the recommedations you will also move that these recommendations be forwarded to the deans of all medical schools as embodying our idea of undergraduate teaching of ophthalmology.

SURVEY OF UNDERGRADUATE OTOLARYNGOLOGY

RALPH A. FENTON, M.D.

PORTLAND, ORE.

If I may be permitted a personal word, I should like to say to the Dean that we owe our present splendid medical school set-up at Portland to the far-sighted vision of a graduate of McGill, and it therefore gives me especial pleasure to have had these words of counsel; we hope he will sit in judgment on our findings here.

Respecting our findings, which you have before you, I think Dr. Gradle's general observations agree rather exactly with those of our committee as to the background of the various schools who answered the questionnaire. Replies were received from 28 of the 80 schools to which questionnaires were sent, some of them complete, and in others it was evident that the heads of the departments were "prima donnas" strutting upon a mimic stage and paying very little attention to the undergraduate students entrusted to them.

In the matter of recommendations, we feel that otolaryngology should receive an independent status and should be budgeted independent of other departments. In pediatrics especially, some schools are having trouble between the head of that department and the department of otolaryngology.

Regarding the question of preliminary courses in anatomy and physiology, with lectures by otolaryngologic chiefs, it is recommended that these subjects be given as a part of the freshman or sophomore instruction. It does not take much time, but the psychologic effect, not only of the lecturers' statements but the broad view it gives the youngsters is thought to be rather wise and has worked out well in departments of anatomy and physiology where it has been tried.

Respecting the number of hours, we have gone into no detail because the content of proper courses is so well known to you; but we feel it should include, as an optimum rather than a maximum or minimum, at least seventy hours, of which sixteen to twenty hours should be junior didactic based upon a standard text. Operative technique should not be taught, and yet it is taught in some places. Recognition of the normal is the important thing, and in certain places recognition of the normal takes up a good deal of time in the junior year, and that is worth while in spite of the fact that these boys are interested in hearing of gross abnormalities. They ought to know the normal before they see the disease.

The recommendation of the committee is that research should not be encouraged among undergraduates except in special cases, and then at the discretion of the research council of the institution concerned, where

special aptitude has been discovered. The suggestion is made that it would be well to have the youngster do such work in a summer term, or when he is taking a five-year course, rather than attempt to carry it along with the tremendous burden of the present courses in surgery, medicine and obstetrics.

We feel elective courses should be encouraged, but limited to advanced work in examination and diagnostic methods. Training in surgical technique should be limited to graduates. The use of films, slides, microscopic projections, should be encouraged in didactic and dispensary work.

Obviously in certain parts of the country it is not possible for junior instructors to appear before the American Board of Otolaryngology in the first two or three years, but we recommend the appointment of no permanent instructors who do not have this certificate.

We adhere also to the view that these findings should be submitted to the deans of medical schools, and we further request that the replies of these gentlemen be referred to the committee, and the committee continued in order that such replies may be codified and a report brought in next year.

DISCUSSION

THE PRESIDENT: Before we have discussion in general I will ask Drs. Jackson, Greenwood, Howard, Barnhill, Newhart and Goldsmith if they will discuss the questions before the meeting.

DR. EDWARD JACKSON, Denver: Mr. President and Fellow Members and Guests of the Academy: The report that has been laid before us in two parts may well be the starting point for what in the end is likely to be an important movement in medicine. The prominence of the two specialties concerned is likely to arouse attention in the profession in general; and on the wisdom of the action that is taken in the direction of these pretty well defined specialties may depend a general movement toward the medicine of the future. I think it is well for us to remember in such a discussion that we are providing for medicine that embraces all that is now known, and looks forward to a more rapid advance, a more rapid increase of knowledge, a more rapid mastery of new methods, than we have ever known before. We have come to a time when the mastery of medicine in its present state of development is not possible to any one mind. No one can master the whole of medicine.

It would be extremely foolish for us to teach undergraduate students that it is possible in four, five, six or seven years in classes, to prepare them for the practice of medicine in general. Medicine, including surgery, can only remain a united profession concerned with the general activities of the community, by giving to all members of the profession certain common education. That that common education all branches of the profession should possess, in order that they may have normal relations with each other, in order that they may understand each other, some knowledge outside of the field of medicine, and together with that some substantial knowledge of their chosen specialty. For all this there must be a common

medical education to give a basis, and that is the problem of all teachers of undergraduate medicine.

It seems an anachronism to speak of preparing general practitioners. All the practitioners of medicine in the undergraduate course should get all the branches of medicine needed in common throughout the profession, and my understanding of the use of such a survey as has been made in the two specialties is that from that we should try to work out a definite conception; not to be put down in doctrine, but to be in the minds of all teachers of these branches, of what is needed by the branches of our profession. The man who ten years from now is practicing pediatrics, or major surgery, or neurology, ought to know certain things about ophthalmology and otolaryngology, or he ought to know how to study them.

A study of our profession, its successes and its failures, should lead us to the decision that certain things ought to be taught. Take one or two instances in ophthalmology. One is the use of the ophthalmoscope. It is an instrument of general diagnosis. It is diagnostic, through the eye, of conditions that may be treated by the experienced general physician or internist, or conditions that may be treated by the surgeon, or by the pediatrician. Every medical student should be so acquainted with the ophthalmoscope, with what it will reveal, that he can resort to the common knowledge of the profession available. That is the reason for taking the ophthalmoscope as a typical diagnostic means of examination, to emphasize the importance it should be given in general diagnosis. One other thing, still speaking of the ophthalmoscope. There is today a lamentable gap between the work done by our highly specialized laboratories, and clinical medicine and surgery. It comes particularly with reference to pathology, from the point of view that specialists do not thoroughly understand what the men they are consulting with think, how they regard the phenomena in question. The ophthalmoscope is in the breach between clinical medicine and the knowledge of pathology that needs to be called to the attention of every medical student. He should see lesions not only under the microscope, but lesions that occur in body as we watch them in successive years. The ophthalmoscope becomes the instrument that shows the lesion under low magnification, where it may be watched from day to day during its progress. With the biomicroscope we may see the same thing. Ophthalmology has to take the position that the eye is the gateway to a large part of medical knowledge, and the student should be trained with the idea that he is preparing to study, in some direction or other; and should have the necessary preparation for the real study of medicine, which begins when he gets his diploma.

Dr. Allen Greenwood, Boston: Mr. President, speaker of the evening, and Ladies and Gentlemen: The teaching of medical students has always been of great interest to me, but I hope we will never get away from the fact that fundamentally the medical schools are to train practitioners of medicine, not specialists. I think medical schools vary in their service to the community. Some are so equipped and have such fine opportunities that they will be likely to turn out men who will eventually specialize along many lines. Other medical schools are more concerned

in the education of men likely to become general practitioners. I do not agree with those who say that the general practitioner is going out. I do not believe this for a minute for such men are badly needed. But he should have the breadth of knowledge that is necessary for a general practitioner, including some ophthalmology. It happens that the first ten years of my medical life were devoted more to general practice and general surgery than to ophthalmology, and it seems to me that I can perhaps see from the standpoint of the general practitioner, and I have the feeling that the man who has done some general practice, or general surgery, thinks more about the general diseases of the patient who comes to him than the man who jumps from the medical school to the practice of his specialty.

What the general practitioner needs of ophthalmology is to learn to recognize and evaluate what he sees. He needs particularly to have his powers of observation trained. The other day in assisting at an American Board examination I saw a patient across the room and asked the men to suggest a diagnosis from what they could see, but only a few observed that the patient had a goiter and a slight exophthalmos. My point is that the medical man must be trained to see everything observable.

Then what does the medical practitioner need of ophthalmology? He should know the normal eye, the normal anatomy, the normal physiology; he needs to know how to tell the commoner external diseases; he needs to be able to use the ophthalmoscope. Medical practitioners often come to me and ask me to give them a course in ophthalmoscopy. I have done it many times. They are grateful for it and they tell me afterwards what a help it is to them in their work.

The general practitioner should be taught the relation of the eye to general diseases. They should know when they see a patient with pinhole pupils what they may look for further; they should be able to pick up many diseases through the eye other than with the ophthalmoscope, but of course the ophthalmoscope will help them in their diagnosis and treatment.

I think the committee has done a wonderful piece of work. I do not know whether sixty hours is wholly necessary in some schools, but in others it is probably not enough. Personally I cannot see the need of a whole hour for cataract. I think they should give most of this hour to some other condition. As I said a minute ago, medical ophthalmology should include a good knowledge of external diseases of the eyes and adnexa, the ability to recognize paralytic squint, and the ability to interpret the findings of the ophthalmologists who have sent patients with a report of ocular findings.

Dr. Harvey J. Howard, St. Louis: Mr. President, Officers, Ladies and Gentlemen: A statement has been made here tonight regarding the importance of training the student to observe. In this connection I recall that one of our great American geologists once gave a student a piece of rock and told him to study it thoroughly and come back and tell him about it. At the end of two days the student went back to the professor believ-

ing that he knew all there was to know about the rock. The professor asked him a few questions, none of which he could answer. He then told the student to study the rock for a month. As the story goes, the student found that a month was not enough and finally after studying it for several months came back to the great geologist in humility and confessed that he was only just beginning to understand the little piece of stone.

I refer to this simple incident in order to stress the importance of careful observation in scientific work. If observation be important in learning to understand inanimate objects, it is probably equally important when it comes to animate objects. Training the student to observe with infinite care and patience is undoubtedly the first duty of the teacher. I cannot conceive of a more pertinent application of an ability to observe accurately than that relating to the study of the eye and its diseases.

This statement introduces the subject of our methods of teaching. The Dean of the Medical School of McGill University stated a fundamental principle when he said tonight that "the student should be largely responsible for his own education." In other words, the student will keep as his own, only that which he digs out for himself. And yet most of our educational methods are built upon a different principle. Although there has been a steady improvement in teaching methods, it is often stated by progressive educationalists that even today our students are spoon-fed through grammar school, and spoon-fed also through high school and through college. When, therefore, they enter medical school most of them expect still to be spoon-fed. And insofar as didactic methods of teaching are maintained, the students continue to get what they expect.

I have had experience in both China and the United States in teaching medical students. I have tried out both the lecture and the case methods of teaching and it is my conclusion that the lecture method, the spoon feeding variety, is largely wasted effort.

Ophthalmology in all of our medical schools is forced to operate on a relatively slender teaching schedule. Any effort on the part of the departments of ophthalmology and otolaryngology to increase their number of required teaching hours has always been met with disfavor by the deans and the executive committees of medical schools. At a time when the general tendency is to cut down on all departmental teaching schedules and give the student more free time, it is hard to blame the deans and committees for their attitude. The only way to justify our position in requesting more teaching hours is to demonstrate that we are using the allotted time we now have so well that the students are enthusiastic about ophthalmology and practically demand more of it. In some schools this demand is met by the offering of elective courses in addition to the required courses in ophthalmology. In all progressive schools there is a more or less constant revision of the medical curriculum. But experience has demonstrated that it is easy to revise a curriculum downward but almost impossible to revise it upward.

There are a few things that I should like to say specifically regarding the undergraduate teaching program presented for our consideration tonight. On the whole I think that the program is very good and that we should commend the committee for the work that they have done as a foundation for their recommendations. I believe that these recommendations should help us a great deal. I suppose, however, that the committee desires a frank discussion of the subject and welcomes friendly criticism. In the first place I am opposed to that part of their schedule which includes so many hours of didactic teaching, a matter to which I have already referred. In the second place I believe that the required curriculum should provide for more than two hours of work in ophthalmoscopy. I suggest that this part of the schedule should be increased to ten or twelve hours and that the extra hours be transferred from the schedule of didactic teaching. In this connection it seems important that the student provide himself with his own ophthalmoscope by the beginning of the third year. In his work on the wards and in the clinics during the last two years of his medical course he will have an opportunity to learn its use and its value. I believe he should always have his ophthalmoscope with him as he does his stethoscope. I know that some ophthalmologists are opposed to this on the basis that the acquisition of a little knowledge is a dangerous thing and that when these students become practitioners of medicine they will do their own fundus work and fail to call in an ophthalmologist for consultation when they should. I cannot agree with such an attitude.

The committee has also made certain recommendations about elective courses in ophthalmology. There is a psychological difficulty connected with such courses in the undergraduate curriculum. In some institutions, as is the case in my own, an elective course is offered in lieu of what should be scheduled as an important required course. It is true that the majority of students take this elective course, but it does stand as a barrier against all of the students getting an important clinical course. I suggest that it might be a better thing for those sponsoring these recommendations in their final form to propose unanimously if possible, a schedule of fifty or sixty hours of required work in ophthalmology and leave out any reference to electives which could be reserved for postgraduate instruction only.

The committee has made a move in the right direction and should have the support of all of us who are interested in teaching ophthalmology.

Dr. John F. Barnhill, Miami Beach, Florida: I agree with much that has been said, but hold to the belief that deans and councils having to do with the arrangement of curricula of medical schools have not always fully appreciated the importance of assigning a reasonable amount of time to teaching ophthalmology and otolaryngology. They seem to cling to the belief that was too true only a few years ago that rhinologists are but sprayers and oilers of the nose and of little consequence.

But what are the present facts? What is the present status of the specialty as relates to medicine as a whole. I will quote the opinion of

others. Evans, University of Wisconsin, believes that a very large number of the most fatal general affections have their origin in focal infections of the head. Billings expresses a similar view. Deland says, "If one wishes to know the secondary effect on the system of focal infections of the accessory nasal sinuses, all he need do is to consult the index of any recent work on the practice of medicine. Most general diseases have such origin." I might quote many others but it is not necessary. The point I wish to make is that the human head, the field of the otolaryngologist, harbors affections which may and do spread to the system where general disorders are set up. Hence the interest to every other specialty in medicine.

To make an adequate study of the origin of systemic disease the sinuses, teeth, tonsils and other structures of the upper respiratory tract cannot be ignored. The questions arise, therefore, "Is any student of medicine properly instructed until he has been well informed concerning the character of the structures, the focal sites which are so often responsible for systemic diseases? Is it possible for any teacher, however well informed and skilled, to instruct the student adequately concerning these foci in the time often alloted?" There are many evidences that this is not possible, and the same is equally true in teaching ophthalmology.

It was long ago said that the eye is the window of the soul. It is more than that; it is also a window through which intracranial changes of vast importance in medicine may be viewed and estimated. A few hours' instruction on what may be seen through this window, and a proper estimation of the changes that may have taken place, are scarcely sufficient. Interpretation in such instances to be worth while can come only after long and patient study with a competent instructor.

But I do not criticize. Medicine as a whole is so enormous that deans must have great difficulty in apportioning time to each department. Moreover, it is not expected of any teacher that he turn out a finished product, a completely informed student. Probably the most that can be expected is that the teacher so instruct the student that he goes on through life learning and completing his medical education long after he has left college. Leaving the school with such an inspiration is perhaps better than too much teaching, and in viewing it thus teachers will usually be satisfied with a reasonable number of hours.

In the field of anatomy, I have the feeling that too many schools give too few hours. I have great respect for the medical man who knows his anatomy, for at the moment I cannot recall a single great physician, surgeon or specialist who was not well versed in anatomy. All the hours that can be spared should be given to teaching this basic, absolutely indispensable subject.

Dr. Horace Newhart, Minneapolis: Mr. President, Dean Martin, Ladies and Gentlemen: I shall limit my remarks to the subject of otology and I ask your indulgence if I am somewhat personal. Twelve years ago in Kansas City it was my privilege to present a paper before this organization on "The Need for More Thorough Training in Otology for Under-

graduates in Medicine." It took a great deal of courage at that time to say some of the things I did. I then expressed the fond hope that at some time in the future some organization would undertake what has been so admirably carried out by this committee. I wish to congratulate the members of the Committee on the fine start they have already made.

In preparing the paper referred to I relied almost entirely upon the statements of courses as they appeared in the bulletins of Class A medical schools. Such statements I have been obliged to conclude do not represent the amount or quality of practical knowledge the student actually acquires. They too often express ideals rather than attainment.

Since the Kansas City meeting it has been my privilege on several occasions to conduct examinations in otology for the National Board of Medical Examiners. The contacts with the men who have come up for examination has been quite a disappointing revelation. As individuals they showed the widest variation in the specific knowledge of the subject they possessed. This could be due chiefly to differences in the amount and quality of the clinical instruction they were supposed to have received. Some did not know how to use the ordinary head mirror and speculum, but insisted on being provided with an electric lighted otoscope before they could even recognize a normal drum head. Some had not even an elementary knowledge of the fundamental functional tests. Others showed a fine practical knowledge of the subject so far as the needs of the general practitioner are concerned, proving that their training had been excellent.

It is our conviction that we owe it to the general public that we insist on an adequate number of hours of undergraduate instruction, both didactic and clinical, so that the general practitioner may be a safe man in his dealings with his patients who consult him for ear diseases. As a minimum requirement he should be able to recognize the commoner ear diseases and realize the possibilities of complications and what should be done to minimize their occurrence. In many schools this today implies an extension of the course by the addition of more hours, including provision for added lectures in the prevention of deafness and its amelioration. This should include the latest methods of detecting hearing loss in the individual and in groups, and the application of modern methods for the prevention of deafness and the best means for the rehabilitation of those who have a handicapping deafness — subjects on which the average general practitioner is far behind the times.

Each general practitioner as he leaves his medical school should be inspired with a solemn sense of the responsibility which he assumes when he undertakes the care of a suppurating ear case. Then he will be less likely to attempt to do work for which he has not had such adequate training, as can be acquired only by a long apprenticeship in otolaryngology.

On one point I differ from the recommendations of the committee: In the overcrowded curriculum for undergraduates I believe there is no time or place for research work.

The suggestion that teachers of clinical otolaryngology be urged and required to hold the certificate of the American Board of Otolaryngology is most commendable.

DR. PERRY G. GOLDSMITH, Toronto: I happen to represent the otolaryngological department of the medical school of Toronto University. I say this so you may not think that McGill is the only medical school in Canada. All the new things we have in teaching gradually find their way to McGill, and if McGill has anything new we find our teachers know something about it.

What is the object of this discussion? I take it this gathering of teachers tonight is to discuss some of the difficulties which arise in the teaching of *undergraduate* students. Surely it is not the object of this meeting to standardize the teaching of otolaryngology. You cannot standardize students or teachers, and certainly not teaching. All medical schools I believe have a committee which meets at various places during the year in order to discuss how best they may adapt new ideas likely to be helpful in securing the best methods of teaching the student to become a *general practitioner*. There are members on that committee who continually lean toward the abolition of the chairs of ophthalmology and otolaryngology, and such medieval ideas have affected the minds of others.

Undergraduate instruction I am perfectly aware cannot be standardized. What is the province of the teacher of undergraduate medicine? Is it not his province to make a well-rounded general practitioner? The State supports McGill and Toronto; the State has an interest in us; she is responsible because legislators are elected by the people who set certain standards of teaching which the universities shall carry out. But what of the public? The public has a right to expect that the new practitioner shall be well versed in the common ailments from which people suffer. If he does not know how to do everything himself they expect him at least to advise them wisely. The rights of the public must not be interfered with, as would occur in unsuitable teaching. Our duty is to see that the confidence and the responsibility given to us by the public is not misplaced.

The duration of teaching — the number of lectures and amount of didactic teaching, varies from 32 to 19 hours. I believe that any teacher who cannot in ten or twelve lectures tell his class of undergraduates all they should be expected to know about otolaryngology has not a clear conception of a general practitioner's work.

So far as books are concerned, when you look at the great volumes the ophthalmologists and otolaryngologists have given us, and recommend in the college calendar, no wonder the student refuses to purchase them. What the poor student wants to know is how to take care of a case, for example, with pain or pus in the ear — and we give him a big book he can hardly lift.

Teaching of undergraduates does not depend on fine buildings, fine libraries, or redundance of equipment. It depends largely on the inspiration the teacher is able to inculcate in the student, in preparing him to

continue studying after he graduates. If the teacher has succeeded, he has given him more than if he mastered all the technical details of unusual operations or the recognition of rare diseases.

Dr. John W. Carmack, Indianapolis: I move that these committees on undergraduate instruction be continued; that an abstract of the reports made by these committees here tonight be sent to the Deans of Class A Medical Schools, and that further reports by these committees be made at our next meeting. (Motion seconded by Dr. Kopetzky, and carried.)

DEVELOPMENT OF POSTGRADUATE QUESTIONNAIRE

DR. S. J. KOPETZKY, New York: In the development of the questionnaire to accumulate information in regard to the status of postgraduate instruction we were confronted with an entirely different proposition from that which confronted the committees whose reports have been offered this evening. Postgraduate instruction comprises, as you know, the further education of the general doctor on the one hand, and on the other the complete development of specialists in our branches of medicine, and the questionnaire should be so constructed as to elicit information as to the ability of postgraduate schools to develop these two types of individuals. The rounding out of the cultural education of the practitioner beyond the medical instruction received with his medical degree rests in large measure upon the graduate instruction in the various branches of medicine, of course including the branches in which we are interested. That is one question.

The other is to take the man who feels called upon to enter the specialties in which we are interested and develop him into a complete specialist, not merely a technician.

For the development of the first group in making up our questionnaire we need information which we can disseminate among the general practitioners; and that should be one portion, in my opinion, of the questionnaire. The other should concern itself with two subdivisions. One, the development of specialists through instruction at institutions equipped to give it; the other, the development into a rounded specialist by residency in an institution rated as a teaching hospital. There is, as we know, a woeful inequality in the manner in which residencies are handled. In many places, unfortunately, the resident in otology is nothing more than the servant of his superiors, or a glorified interne, and the question of his education never is in his own hands. He has been misled into accepting the residency in the hope that under the inspiration of those above him he will be taught how to study, how to work to round himself out. An inquiry should be made in such a manner as to uncover the good places and recommend them to such as seek development through residencies.

In regard to institutional graduate teaching, a questionnaire should be developed to determine by analysis, as these committees have done, what is given of the fundamentals, what is given of the clinical side, how much of surgical technique is actually given the graduate student, and what research work is going on through which the man eventually develops. There are men who are highly respected in our Academy who are very one-sided in their development because of their trend toward research, and they do not become the good all-round practical specialists of use at

the bedside. Their pre-eminence in a particular sphere of research should not be the lure which brings men to them for development as all-around specialists when they, the teachers, are hardly in that class themselves.

Thus you see, gentlemen, that it is a harder problem, in a way, to formulate a questionnaire for the graduate department than for the undergraduate department. Medical schools are principally interested in conferring the medical degree, although in many instances the deans may have as an addition an extension course. But how many of these courses constitute a separate, distinct department under a separate dean? How many are combined with the undergraduate department? I would suggest that we empower this committee to get up a questionnaire, to analyze the replies and present that analysis at next year's meeting of this Teachers' Section, and then have a full and free discussion, and after that send the matured opinion of this Section to the deans concerned with the post-graduate and graduate instruction, with the idea of acting in an advisory capacity to them. Likewise, any matured opinions which the report may develop should be sent to the governing authorities of these teaching hospitals who maintain residencies in our specialties, with the same purpose in mind.

It has not been brought out in the discussion here this evening, but the members of these committees have told me that the deans of undergraduate schools have shown happiness at receiving suggestions from the committee which studies the situation in regard to undergraduates. Whether or not the deans can act on these suggestions is not always in their own hands, but they have been glad to receive the suggestions. I think if the graduate part of the problem is handled similarly that the deans dealing with the problem of graduate education will likewise be happy to receive our reports and our mature judgment and suggestions.

DR. W. P. WHERRY, Omaha: I would like to move that the President and the President-elect get together and appoint a Postgraduate Committee, empowering them to develop a standard procedure and to report their progress at our next meeting. (Motion seconded by Dr. Kopetzky and carried.)

The Teachers' Section adjourned *sine die*.

INSTRUCTIONAL SUBJECTS

Special Courses

1. "HISTOPATHOLOGY OF THE EYE"

   Dr. J. S. Friedenwald, Baltimore, Md.
   Dr. R. C. Gamble, Chicago, Ill.
   Dr. W. E. Camp, Minneapolis, Minn.
   Dr. Georgiana Dvorak Theobald, Chicago, Ill.

2. "HISTOPATHOLOGY OF THE NOSE, THROAT AND EAR"

   Dr. Joseph C. Beck, Chicago, Ill.
   Dr. LeRoy A. Schall, Boston, Mass.
   Dr. Gregor McGregor, Montreal, Canada
   Dr. M. Reese Guttman, Chicago, Ill.
   Miss Dorothy Wolff, St. Louis, Mo.

3. "LESIONS OF THE FUNDUS"

   Dr. Arthur J. Bedell, Albany, N. Y.

   Acute Inflammatory Lesions
   Vascular Lesions
   Degenerative Lesions

4. "CLINICAL PATHOLOGY OF OTITIC LESIONS"

   Dr. Samuel J. Kopetzky, New York, N. Y.

   Clinical Pathology of Acute and Chronic Middle Ear Infections
   Clinical Pathology of Otitic Systemic Infections
   Clinical Pathology of Complicating Intracranial Lesions

Otolaryngology

DR. RALPH W. ALMOUR, New York, N. Y.
The Surgical Attack of Empyema of the Petrous Apex

DR. JAMES A. BABBITT, Philadelphia, Pa.
Tinnitus Aurium and Vertigo

DR. LOUIS J. BIRSNER, St. Louis, Mo.
Anatomical Demonstration

MR. EDGAR B. BURCHELL, New York, N. Y.
The Temporal Bone and Its Surgical Anatomy and Abnormalities

DR. JOHN W. CARMACK, Indianapolis, Ind.
Acute Otitis Media With Various Complications

DR. JAMES B. COSTEN, St. Louis, Mo.
Surgical Anatomy of Some Procedures in Otolaryngology

DR. RALPH A. FENTON, Portland, Oregon
The Conservative Treatment of Chronic Middle Ear Suppuration

DR. THOMAS C. GALLOWAY, Evanston, Ill.
The Use and Limitations of Surgical Diathermy

DR. BERT E. HEMPSTEAD, Rochester, Minn.
Diagnosis and Surgery of Ethmoid and Sphenoid

DR. MARVIN F. JONES, New York, N. Y.
Phases of Neurology of Importance to the Head Specialties

DR. V. H. KAZANJIAN, Boston, Mass.
Nasal Tip Deformities and Other Plastic Procedures on the Nose

DR. A. S. MacMILLAN, Boston, Mass.
X-Ray Examination of the Accessory Sinuses and Mastoids

DR. C. F. McCLINTIC, Detroit, Mich.
Headaches

DR. J. B. McMURRAY, Washington, Pa.
Evaluation of Nose and Throat Conditions as They Involve the Eye

DR. WILLIAM MITHOEFER, Cincinnati, Ohio
Method of Treating the Voice Defect of the Singer and Public Speaker

DR. HORACE NEWHART, Minneapolis, Minn.
The Practical Use of the Audiometer

DR. FRANK J. NOVAK, JR., Chicago, Ill.
The Practical Application of the Newer Knowledge of Immunity to Otolaryngology

DR. HENRY B. ORTON, Newark, N. J.
Plaster Demonstration of Laryngectomy and Trans-Thyroid Pharyngectomy

DR. LYMAN G. RICHARDS, Boston, Mass.
Diagnosis and Treatment of Sinus Disease in Children

DR. ROBERT F. RIDPATH, Philadelphia, Pa.
The Lateral Nasal Wall

DR. JOHN J. SHEA, Memphis, Tenn.
Helping the Sinus Patient

DR. BENJAMIN H. SHUSTER, Philadelphia, Pa.
Labyrinthine Diagnosis

DR. DUNCAN CAMPBELL SMYTH, Boston, Mass.
Bronchoscopy as an Aid in the Diagnosis and Treatment of Diseases of the Lungs

DR. ROBERT SONNENSCHEIN, Chicago, Ill.
Reflex or Referred Signs and Symptoms of Diseases of the Nose, Throat and Ear—Such as Sneezing, Coughing, Vomiting, Pain, etc.

DR. GABRIEL TUCKER, Philadelphia, Pa.
Cancer of the Larynx

DR. SAMUEL E. WHITNALL, Montreal, Canada
Some Points in the Anatomy of the Orbit

Ophthalmology

DR. FRANCIS HEED ADLER, Philadelphia, Pa.
Herpes of the Eye and Adnexa

DR. S. JUDD BEACH, Portland, Maine
Refinements of Refraction

DR. CONRAD BERENS, New York, N. Y.
Ophthalmic Phase of Aviation Examination

DR. MELVILLE BLACK, Denver, Colo.
Ocular Complications of Infectious Diseases in Children

DR. NELSON MILES BLACK, Miami, Fla.
Compensation in Industrial Ophthalmology

DR. ALBERT L. BROWN, Cincinnati, Ohio
Non-Surgical Treatment of Diseases of the Tearsac

(Through courtesy of Dr. Edmund B. Spaeth, a moving picture film of an extirpation of the tearsac, showing the regional anatomy and the surgical landmarks was presented.)

DR. G. E. CLAY, Atlanta, Ga.
Ophthalmic Neurology

DR. MARTIN COHEN, New York, N. Y.
The Management of Simple Glaucoma

DR. ALFRED COWAN, Philadelphia, Pa.
Localization of Retinal Tears in Detachment

DR. W. THORNWALL DAVIS, Washington, D. C.
Military Ophthalmology

DR. J. H. DUNNINGTON, New York, N. Y.
Operative Treatment of Squint in Children

DR. ED. C. ELLETT, Memphis, Tenn.
Operations Upon the Iris for Glaucoma

DR. J. N. EVANS, Brooklyn, N. Y.
The Interpretation of Central Field Defects

DR. EARL B. FOWLER, Chicago, Ill.
Practical Cylinder Retinoscopy

DR. D. F. GILLETTE, Syracuse, N. Y.
Vitreous Opacities — Etiology, Diagnosis, Management

DR. J. F. GIPNER, Rochester, N. Y.
Use of Modern Optical Instruments of Precision

DR. EVERETT L. GOAR, Houston, Texas
Tumors About the Eyeball

DR. D. B. KIRBY, New York, N. Y.
Ptosis — Its Cause and Correction

DR. PETER C. KRONFELD, Chicago, Ill.
Clinical Application of Some Modern Research

DR. A. N. LEMOINE, Kansas City, Mo.
Manifestations of Allergy in Ophthalmology

DR. F. PARK LEWIS, Buffalo, N. Y.
Sight-Saving Classes in the Schools

DR. RALPH I. LLOYD, Brooklyn, N. Y.
Diagnosis of the Important, Unusual Fundus Conditions

DR. ALEXANDER E. MacDONALD, Toronto, Canada
Optic Neuritis — Diagnosis, Etiology, Pathology

DR. R. J. MASTERS, Indianapolis, Ind.
Radium and Roentgen-Ray Therapy in Ophthalmology

DR. JOSEPH L. McCOOL, San Francisco, Calif.
The O'Connor Operation for Correction of Squint

DR. PAUL G. MOORE, Cleveland, Ohio
Traumatic Surgery of the Eye

DR. C. S. O'BRIEN, Iowa City, Iowa
Intracapsular Cataract Extraction

DR. ALBERT D. RUEDEMANN, Cleveland, Ohio
Exophthalmos

DR. BERNARD SAMUELS, New York, N. Y.
Histopathology of Corneal Disease

DR. MARK J. SCHOENBERG, New York, N. Y.
Management of Acute Inflammatory Glaucoma

DR. EDMUND B. SPAETH, Philadelphia, Pa.
Free Skin Grafts

DR. E. STIEREN, Pittsburgh, Pa.
Management of Intraocular Foreign Bodies

DR. DAVID W. WELLS, Boston, Mass.
Stereoscopic Treatment of Heterophoria

DR. M. F. WEYMANN, Los Angeles, Calif.
Commoner Infections of the Conjunctiva

PRESENTATION OF INSTRUMENTS AND APPLIANCES

Section on Ophthalmology

A NEW BED FOR HEAD CASES *

HARVEY J. HOWARD, M.D.

ST. LOUIS, MO.

FIG. 1—The bed with the head end in position with friction handles closed. Detachable irrigating rod with a socket on each head post. The cranks at the foot of the bed permit the placing of the patient in several positions.

* Manufactured by The Simmons Company.

THE ORDINARY hospital bed does not permit the surgeon to stand at the head of the bed in dressing cases involving the eye, ear, nose and throat, and the brain. As a result of the need for something different, I present today a bed specially adapted for the dressing of all types of head cases. The principal feature of the bed is a hinged head, which can be folded backward by releasing two easily manipulated friction handles on the inner sides of the bed posts. The operation is made noiseless and smooth by the use of properly placed rubber bumpers. The head of the bed is somewhat lower than beds generally used, but is high enough to hold two pillows in place when the headpiece is in position. An important application of the bed is that it is a potential operating table.

In addition to the special hinged-head feature the bed is supplied with a detachable irrigating rod with a socket for it on

FIG. 2—The bed with the head-piece at the level of the mattress folded backward by releasing the two friction handles.

each post. The bed also possesses all the other newest features of the best hospital beds.

A study of the three illustrations and their legends will explain the special features and their action more in detail.

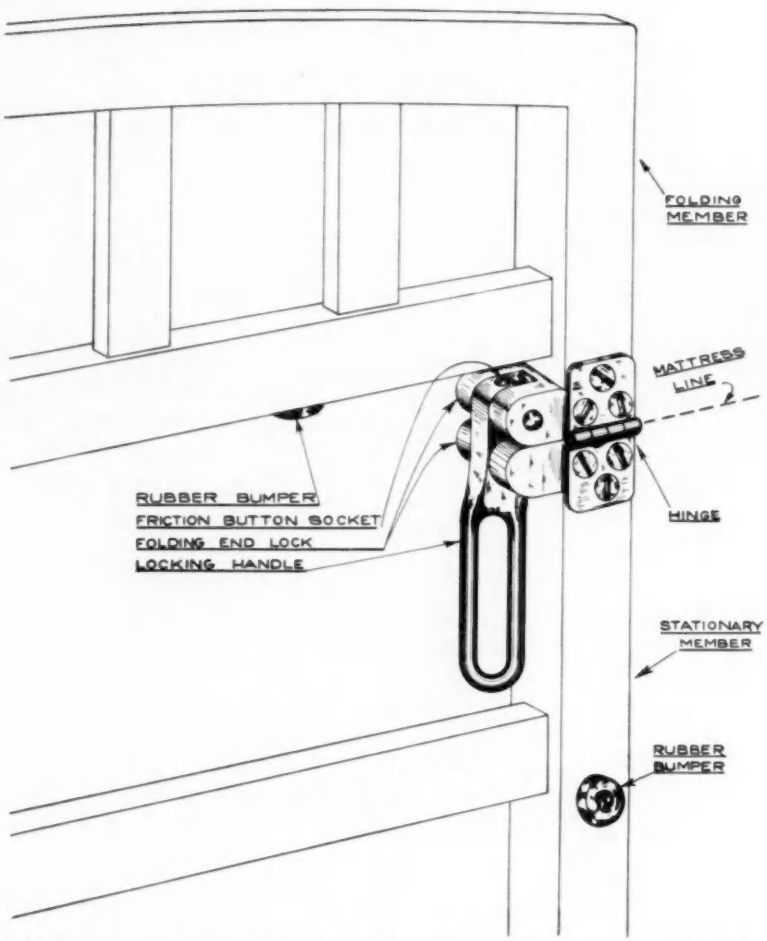


FIG. 3—A diagram of the hinge and the friction handles from the rear right side.

PRESSED CELLULOID SHIELDS FOR EYE OPERATIONS *

FRANK E. BURCH, M.D.

ST. PAUL, MINN.

THESE NEW celluloid Guild Shields are made of pressed celluloid, and are both double and single. They may be used after any operative procedure, but were especially designed for cataract and ptosis operations. They comprise the following features:

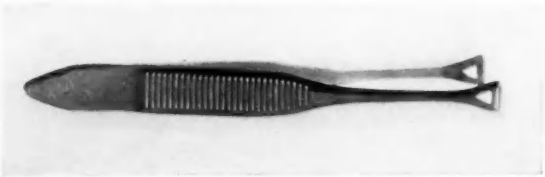
1. They are light and not as cumbersome as the Fuchs' mask, yet provide ample protection.
2. They are both transparent and black.
3. They permit inspection without removal of the mask when open dressings are desired, especially after bridge flap cataract operations.
4. The non-perforated mask may be used for ptosis operations to prevent corneal drying.

* Made by Arthur F. Williams, Guild Optician, 366 St. Peter St., St. Paul, Minn.

INTRAOCULAR FORCEPS

EDWARD STIEREN, M.D.

PITTSBURGH, PA.



I HAVE DESIGNED a pair of forceps to grasp non-magnetic foreign bodies within the globe, the chief feature being the jaws, which are thin, deeply-serrated and fenestrated with triangular openings.

With these forceps, any object, irrespective of its shape, can be grasped and removed from the eyeball. They will hold shot as firmly as irregularly-shaped foreign bodies of copper, glass, stone or wood.

The illustration shows the actual size of the instrument and its light construction. The edges of the jaws are beveled to 0.25 m.m. and are but 0.5 m.m. in thickness, thus allowing their introduction with a minimum of trauma. The spring handles are unusually rigid to provide at all times perfect apposition of the serrated faces of the jaws.

The instrument was made for me by V. Mueller and Co.

A NEW TENOTOMY HOOK AND LID RETRACTOR*

W. D. Rowland, M.D.

BOSTON, MASS.

This hook has an offset to slip under the tendon or muscle after it has been picked up in the usual way, and as it rests in the horizontal position it remains stable because of the broad triangular handle.

It was designed to obviate the difficulty of keeping a muscle hook in place. The classic hook, as you all know, readily and almost always disengages itself when released by the surgeon or assistants, and not infrequently falls to the floor. Once inserted under the muscle, this hook remains there, allowing other bimanual procedures by the surgeon, often desirable.

It was found also to be quite a satisfactory lid hook, fully retracting the upper lid to the orbital wall, and slipping readily to any side position for needed elevation, as often arises. It is easily and tirelessly held in position and is not bulky.

* Made by Mueller and Co., Chicago, Illinois.

PRESENTATION OF INSTRUMENTS AND APPLIANCES

Section on Otolaryngology

THE POINTMETER

EMANUEL M. JOSEPHSON, M.D.

NEW YORK, N. Y.

IN THE STUDY of vestibular reactions there has been a tendency to study the reaction to irritative stimuli and to utterly disregard the responses to normal stimuli. Among the reactions which have come to be abandoned as unreliable are the pointing and past-pointing reactions. My experience indicates that the only possible explanation for the failure of realization of the value of the pointing reactions is lack of adequate study of them.

One of the first facts to be borne in mind in the testing of the pointing reactions in the normal is the fact, as was pointed out by myself in 1923 and Fisher in 1924, that there exists an oculo-vestibular element as well as a vestibulo-ocular element in that summation of impulses which constitute the static and kinetic senses in the human. This was demonstrated by a number of experiments in the lower vertebrates by earlier observers and was more recently embodied in the so-called umbrella test. The significance of this reaction in the presence of an intact vestibular apparatus in the human is that rotation of the eyes to the side of the pointing arm occasions past-pointing. This reaction does not occur when the function of the vestibular apparatus is depressed. In the presence of paresis or paralysis of the eye muscles, pointing is likewise affected, as was determined by von Graefe many decades ago, and by him described as a test for those conditions.

An even more significant reaction from the point of view of the clinician is the head-neck past-pointing reaction. This reaction is the equivalent in the human of postural changes de-

scribed by Magnus and de Kleyn in their experiments on the lower vertebrates. In the normal human there can be elicited no past-pointing on turning the head to either side. In the presence of lesions of the brain, however, past-pointing may be elicited on rotation of the head. Thus far, I have only found this sign in the presence of lesions involving or pressing upon the medulla. I have found it to be quite reliable as an index of such lesions.

There remains much to be learned about the pointing reactions. The pointmeter which I have designed and here present is devised for the purpose of measuring the deviations in pointing in all planes. One thing should be borne in mind in the use of this apparatus, and in all pointing tests: Do not ask the patient to touch the finger or pointer when he has shut his eyes after raising or dropping his extremity; but ask the patient "point to where you think the pointer (or finger) is." In so doing one gets a better picture of the patient's sense of position than one does when the patient attempts to feel his way to the object to which he is pointing.

THERMOS IRRIGATING BOTTLE BY AIR PRESSURE*

WALTER H. THEOBALD, M.D.

CHICAGO, ILL.

I AM PRESENTING this irrigating thermos bottle with some modesty. It has been in use for ten or twelve years, and at the request of some of our colleagues and because of the simplicity and practicability of it, I am presenting it to you. Any solution for irrigating purposes may be used. The warm temperature of the fluid employed at the time of filling the thermos bottle is maintained for six to eight hours. This obviates the necessity of preparing fresh solution every time irrigation of a sinus is required. The use of rubber bulbs and syringes may be dispensed

* Made by Mueller and Co., Chicago, Illinois.

with. Whether your pressure is twenty of fifty pounds does not make any difference because at the top of the vacuum bottle is a blow-off valve which regulates the pressure of stream used. This can be connected with a trocar, or any canula used for irrigating purposes, as the case may be.

The thermos irrigator consists of a one-half gallon bottle with stainless steel filler, this being desirable on account of the pressure which is used. To this bottle is fitted an expanding stopper which is adjustable by means of a wing-screw through which passes the air inlet tube for pressure and the outlet for solution.

An adjustable blow-off valve is provided so that the air pressure may be held in check at the right point to permit an even flow of the solution at low pressure.

The line pressure of compressed air will naturally vary at times, but with this valve the safety of the pressure on the solution is maintained in the neighborhood of ten to twelve pounds.

Both inlet and outlet fittings are provided with a tapered bayonet attachment to facilitate the easy removing of the container from the wall bracket in which it is generally mounted. This same type of fitting is used for attaching irrigating tubes.

A NEW AUDIOGRAM BLANK

HORACE NEWHART, M.D.

MINNEAPOLIS, MINN.

WE PRESENT a new form of audiogram blank for use with the 2-A audiometer. It possesses the advantage of greater compactness than the larger forms heretofore supplied by the Western Electric Company, these being four by six inches in size, which makes it more convenient for filing. The size is also more easily enclosed with letters reporting our findings to physicians referring cases to us.

It has the further advantage of having the numbers indicating the frequency at the top of the sheet thus facilitating the recording of the hearing loss on each perpendicular line, which on the old form has been rendered difficult because the number indicating the line showing the pitch was obscured by the writer's hand.

The new form suggests a uniform method of recording the findings for the right and left ear, which heretofore has been

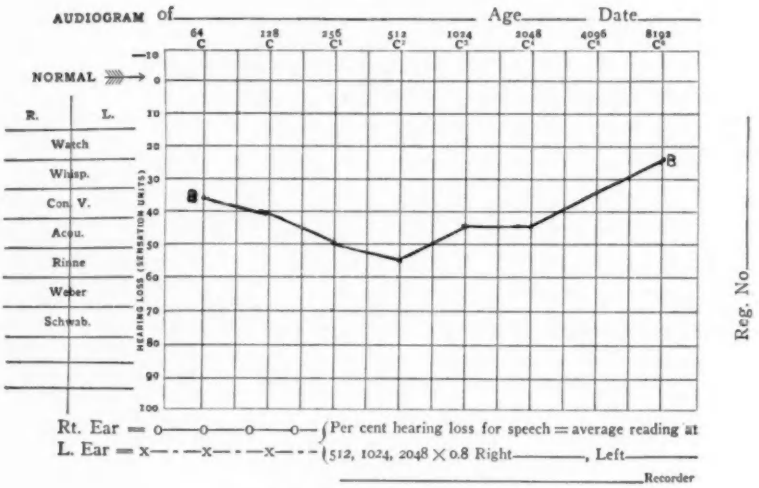


FIG. 1—Shows the new form.

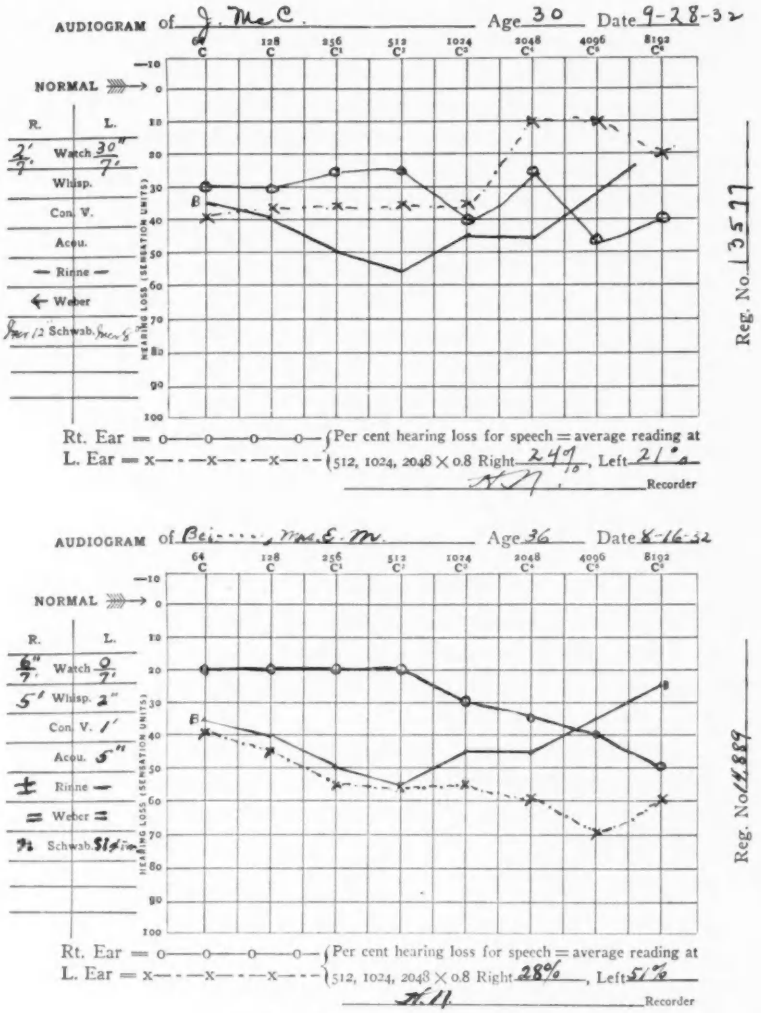

AUDIOGRAM of J. Mc C. Age 30 Date 9-28-32
64 C 128 C 256 C1 512 C2 1024 C3 2048 C4 4096 C5 8192 C6
NORMAL
R. L.
Watch 30"
Whisp.
Con. V.
Acou.
Rinne
Weber
Schwab.
HEARING LOSS (SENSATION UNITS)
Rt. Ear = o—o—o—o—o—
L. Ear = x—x—x—x—x—
Per cent hearing loss for speech = average reading at 512, 1024, 2048 × 0.8 Right 24%, Left 21%
Recorder
Reg. No. 13517
AUDIOGRAM of Bei...., Mrs. E. M. Age 36 Date 8-16-32
64 C 128 C 256 C1 512 C2 1024 C3 2048 C4 4096 C5 8192 C6
NORMAL
R. L.
Watch
Whisp. 2"
Con. V. 1'
Acou. 5"
Rinne
Weber
Schwab.
HEARING LOSS (SENSATION UNITS)
Rt. Ear = o—o—o—o—o—
L. Ear = x—x—x—x—x—
Per cent hearing loss for speech = average reading at 512, 1024, 2048 × 0.8 Right 28%, Left 51%
Recorder
Reg. No. 11889

FIG. 2—Shows the completed audiogram.

left to choice. Many physicians we note are still using various colors to differentiate between the right and left ear. It is our practice to use black for air conduction and colors for bone conduction.

At the left of the sheet we have provided space to briefly record such of the fundamental tests as may be made at the same time the audiometer is used.

We have been told the Western Electric Company will supply blanks of this type in the near future.

A CORE DRILL (TREPHINE) FOR THE POSTMORTEM EXPOSURE OF THE ACCESSORY NASAL SINUSES, POSTERIOR PHARYNX, AND LARYNX

F. Lowell Dunn, M.D., and D. Rhea

Department of Clinical Research

University of Nebraska College of Medicine

OMAHA, NEBR.

Presented by W. P. Wherry, M.D.

The lack of adequate postmortem studies of the accessory nasal sinuses and contiguous structures can, in part, be explained by the technical difficulties in exposing this region. The studies of Harke in 1895 indicate the urgent need of such studies. He found 135 instances of gross inflammation of the paranasal sinuses in 399 autopsies. The study of surgically-removed specimens, which is occasionally done, does not provide the more complete information obtained by postmortem studies. The possible bearing of nasal infections on a large group of diseases must remain largely a matter of clinical opinion until adequate postmortem examinations have been made.

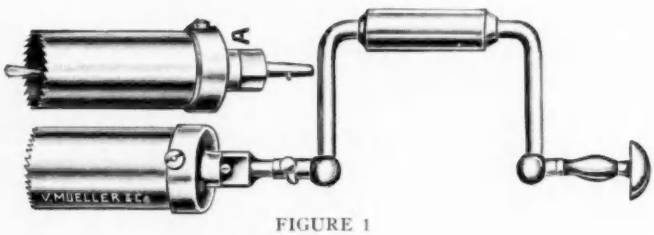


FIGURE 1

A satisfactory method for postmortem study must give adequate exposure in a minimum time and must not introduce any unfamiliar embalming problems. The Harke sagittal head splitting technique and exposure of this region from the face have these objections. A core drill (Fig. 1) has been developed which permits exposure through the floor of the cranial cavity. The drill illustrated is two inches in diameter. After removal

of the calvarium and brain the center drill is put in the pituitary fossa. The axis of drilling is in the midsagittal plane and about midway between the tip of the mandible and the angle. The notches of the facial artery furnish useful guides. After chattering for a few revolutions the drill cuts rapidly into the postnasal pharynx and the core is then held only by soft tissues. The core is removed by traction; however, it is made of sufficiently heavy tubing (one-sixteenth inch) so that sufficient bone is removed to permit sliding a thin-bladed knife between core and wall so that the soft tissue can be cut. The core removed is illustrated in Figure 2. It has been sawed open in the mid-

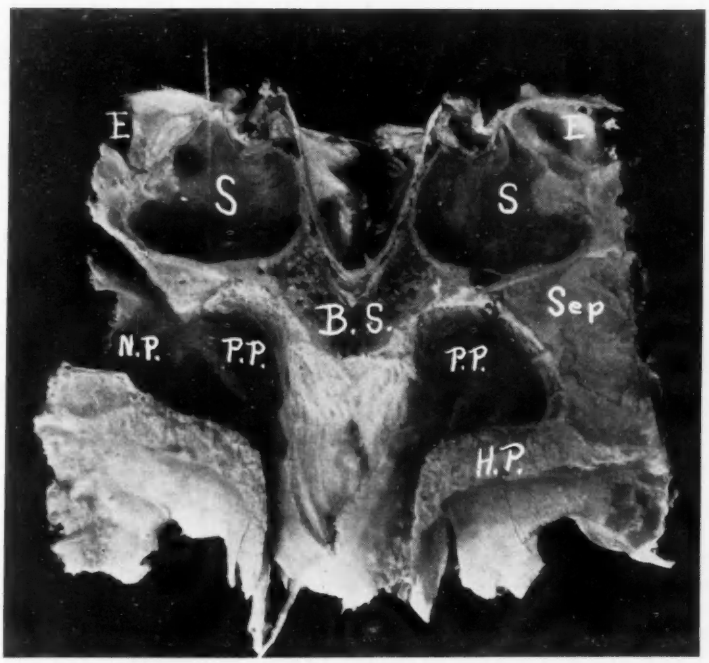


FIGURE 2

sagittal plane. The excellent exposure of sphenoid and posterior ethmoid sinuses, postnasal pharynx, septum, hard and soft palates, and posterior turbinates is apparent. The drill is of sufficient size so that the posteromesial walls of the maxillary sinuses are removed, permitting their examination. The tongue is com-

pletely exposed and the tonsils can be easily removed. Pressure on the base of the tongue exposes the pharynx and larynx. The exposure of the remaining ethmoids and turbinates can be done with the rongeur.

The method described does not introduce any new embalming problem. The core can be removed in five to ten minutes.

REFERENCES

1. Paranasal Sinusitis. A Problem in General Medical Practice. A. D. Dunn, M.D., and F. Lowell Dunn, M.D., Annals of Internal Medicine, pages 235-252, Vol. VI, 1932.
2. A Core Drill (Trephine) for the Postmortem Exposure of the Accessory Nasal Sinuses, Nasal Passages, Mouth and Pharynx. F. Lowell Dunn, M.D., and D. Rhea. Archives of Pathology, 1933.

MINUTES

AMERICAN ACADEMY *of* OPHTHALMOLOGY *and* OTOLARYNGOLOGY

Joint Session

THE JOINT SESSION convened at nine o'clock, Tuesday morning, September 20th, in the convention hall of the Mount Royal Hotel, Montreal, the President, Dr. S. Hanford McKee of Montreal, presiding.

THE PRESIDENT: The Academy indeed is very fortunate in having the Mayor of Montreal with us. May I express to you, sir, the appreciation of the Academy in your presence with us this morning. It is with pleasure I introduce the Hon. Fernand Rinfret, Mayor of Montreal.

HON. FERNAND RINFRET: Mr. President, and Gentlemen: It is a great pleasure and a great honor for me as Mayor of Montreal to welcome you to our great city, and I desire in my first words to thank you most cordially for having chosen Montreal as the meeting place of your convention. I am very grateful to your President, Dr. McKee, for what he has said. I congratulate Dr. McKee on his election to your presidency. I understand this is the first time that such an honor has come to Canada, and I join with the other citizens of Canada in general and Montreal in particular, in expressing our gratitude to the Academy for having made such an excellent selection.

I welcome the members of the American Academy of Ophthalmology and Otolaryngology (I beg you to excuse me if I do not pronounce these difficult words as they should be), and I hope your convention in Montreal proves to be a complete success. I have had the privilege already of meeting some of your convention people. I have reviewed your program, inspected the convention setup, and admit I was somewhat astonished at the large number of instruments exhibited. My first impression while looking at them was to congratulate myself on having avoided thus far their contact with my person. I realize though one must be cautious in speaking of such things, since when one

is ill the first thought is to send for a doctor and his tools. Therefore, I will avoid further thought upon this subject, leaving for you during the course of the convention to deal properly with these scientific matters.

Again I say that I hope you will find Montreal all you expect it to be. I would like to see you make this city your permanent meeting place. We greet our American friends from the United States with open arms. We anticipate their visit and enjoy it when they come, and I am sure that true Americans will always feel perfectly at home when visiting Canada.

I have been asked to express our regret at the absence of him who was supposed to be your guest of honor, Lt. Col. R. E. Wright, a Montreal native. As you are aware, he has been held in England on account of illness. We are all very sorry, indeed, that such a distinguished visitor as Colonel Wright has not been able to complete his journey to Montreal, and as Mayor of the city, may I be permitted to express regret, both of the city and yourselves for his absence?

I thank you for having asked me to open your convention. I hope it will prove very profitable. We are all greatly interested in the development and evolution of medical science. We feel it is at the bottom of the prosperity of society, and that especially in this depression the greatest wealth anybody can have at his command is good health.

We are really interested in your work, and that is the reason I do not feel I should delay you any longer. Again I say that you are welcome to Montreal, whether you come individually or to attend a convention. As Mayor I am very proud this morning and in the name of our citizens I say that we feel honored that the American Academy of Ophthalmology and Otolaryngology is meeting within our walls.

CONVENTION ANNOUNCEMENTS

GEN. H. S. BIRKETT: Mr. President and Fellows: I have the following announcements to make.

First, the President's luncheon. The President, Dr. McKee, is entertaining at luncheon today those who appear on the program, both scientific and instructional, at twelve-thirty in the Salle Dorée on the ground floor.

Second, the Teachers' Section dinner will be held tonight at seven o'clock in the same place. Tickets are to be obtained at the registration desk. The banquet tickets can also be had at the registration desk. I would urge upon everyone to get these tickets early, so the hotel people may be informed how many will be present.

Those Fellows who are Rotarians are reminded of the fact that the Rotary Club is holding a luncheon at the Windsor Hotel at 12:45 today, to which, of course, they are cordially invited.

The Committee would impress upon each and every one of you to leave your railroad tickets at the Registration Desk at once. This is absolutely necessary in order that those who have travelled here may have the reduced rate returning.

You perhaps are all aware that the late Sir William Osler bequeathed to McGill University Medical Faculty the library which he spent the greater part of his life collecting. This was left as a gift to the medical faculty. His nephew, Dr. Francis, is in charge of that library, and it is open for your inspection and interest, and in order to add to that interest Dr. Francis has on exhibition many of the valuable treasures of Dr. Osler where you may be able to study them at any time, I am sure with pleasure and profit. The Medical Faculty Building of McGill is easily accessible — about ten minutes' walk.

This concludes the announcements I have to make at this time.

SYMPOSIUM

The following symposium on "The Progress of General Medicine in Relation to Eye, Ear, Nose and Throat" was presented:

"Blood Dyscrasias in Relation to Diseases of the Eye, Ear, Nose and Throat," Dr. O. H. Perry Pepper, Philadelphia, Pa.

"Cardio-Vascular Diseases in Relation to the Eye," Dr. Norman M. Keith, Rochester, Minnesota.

"The Progress of Medical Neurology in Relation to Diseases of the Eye, Ear, Nose and Throat," Dr. Wilder Penfield, Montreal, Canada.

"Metabolic Studies of Diseases of the Eyes, Ears, Nose and Throat," Dr. I. M. Rabinowitch, Montreal, Canada.

Summary and Discussion, Dr. Perry G. Goldsmith, Toronto, Canada.

The Address of the President was read by Dr. S. Hanford McKee, Montreal, Canada, Dr. Gordon B. New in the chair.

The Tuesday morning session adjourned.

TUESDAY, SEPTEMBER 20, 2:00 P.M.

Section on Otolaryngology

The first meeting of the Section on Otolaryngology convened at two o'clock, Tuesday afternoon, September 20th, Dr. Gordon B. New in the chair.

Dr. Ralph W. Almour, New York, read a paper entitled "The Practical Application of Wittmaack's Theory of Pneumatization." Discussed by Drs. James A. Babbitt, Philadelphia; E. M. Josephson, New York, and Ralph W. Almour.

Dr. L. M. Polvogt, Baltimore, read a paper (written in conjunction with Drs. S. J. Crowe and Stacy R. Guild) entitled "Difficulties of Interpretation of the Audiometer and Other Hearing Tests." Discussed by Drs. Horace Newhart, Minneapolis; Robert Sonnenschein, Chicago; E. M. Josephson, New York, and L. M. Polvogt.

Dr. Louis H. Clerf, Philadelphia, read a paper entitled "Cicatricial Stenosis of the Larynx." Discussed by Drs. Henry B. Orton, Newark, N. J.; E. G. Gill, Roanoke, Va., and Louis H. Clerf.

Dr. August L. Beck, New Rochelle, N. Y., read a paper entitled "A Study of Twenty-Four Cases of Neck Infection." Discussed by Drs. T. E. Carmody, Denver; Harris P. Mosher, Boston; John F. Barnhill, Miami Beach, Fla.; T. C. Galloway, Evanston, Ill., and August L. Beck.

The Tuesday afternoon session adjourned.

TUESDAY, SEPTEMBER 20, 7:00 P.M.

Teachers' Section

The Teachers' Section held its usual dinner meeting Tuesday evening, the President, Dr. S. Hanford McKee of Montreal, presiding. The following program was presented:

"Modern Trend of Undergraduate Medical Teaching," Dr. C. F. Martin, Dean, McGill University, Montreal.

"Survey of Undergraduate Ophthalmology," Dr. Harry S. Gradle, Chicago.

"Survey of Undergraduate Otolaryngology," Dr. Ralph A. Fenton, Portland, Oregon.

These two reports were discussed by Drs. Edward Jackson, Denver; Allen Greenwood, Boston; Harvey J. Howard, St. Louis; John F. Barnhill, Miami Beach, Fla.; Horace Newhart, Minneapolis, and Perry G. Goldsmith, Toronto.

Dr. T. E. Carmody: I move that these committees on undergraduate instruction be continued; that an abstract of the reports made by these committees here tonight be sent to the Deans of Class A Medical Schools, and that further reports by these committees be made at our next meeting. (Motion seconded by Dr. Kopetzky, and carried).

"Development of Postgraduate Questionnaire," Dr. S. J. Kopetzky, New York.

Dr. W. P. Wherry: I would like to move that the President and the President-Elect appoint a Postgraduate Committee, empowering them to develop a standard procedure and to report their progress at our next meeting. (Motion seconded by Dr. Kopetzky and carried).

The Teachers' Section adjourned.

WEDNESDAY, SEPTEMBER 21, 2:00 P.M.

Section on Ophthalmology

The Wednesday afternoon session convened at two o'clock, Dr. S. Hanford McKee presiding.

Dr. Harvey J. Howard, St. Louis, read a paper entitled "Recent Developments in Trachoma Investigations." Discussed by Drs. Martin Cohen, New York; S. B. Muncaster, Washington, D. C.; Sanford R. Gifford, Chicago; S. Hanford McKee, Montreal, and Harvey J. Howard.

Dr. W. Thornwall Davis, Washington, D. C., read a paper entitled "Superficial Parenchymatous Punctate Keratitis." Discussed by Drs. Walter E. Camp, Minneapolis; Oscar Wilkinson, Washington, D. C., and W. Thornwall Davis.

Dr. Parker Heath, Detroit, read a paper entitled "Ocular Lipid Histiocytosis and Allied Storage Phenomena." Discussed by Dr. C. S. O'Brien, Iowa City, Iowa.

Dr. Daniel B. Kirby, New York, made a report: "Study of Disturbances of Carbohydrate Metabolism in Relation to Cataract." No discussion.

Dr. Alfred F. Luhr and Dr. John L. Eckel, Buffalo, presented a paper entitled "Fixation Nystagmus—Report of Cases." Discussed by Drs. John H. Dunnington, New York; E. E. Blaauw, Buffalo; Edward Jackson, Denver, and Alfred F. Luhr.

The Wednesday afternoon session adjourned.

THURSDAY, SEPTEMBER 22, 2:00 P.M.

Section on Otolaryngology

The Thursday afternoon session convened at two o'clock, Dr. Gordon B. New presiding.

Presentation of New Instruments:

Dr. E. M. Josephson, New York, presented a Pointmeter.

Dr. W. H. Theobold, Chicago, presented an irrigating bottle.

Dr. Horace Newhart, Minneapolis, presented an improved audiometer blank.

Dr. W. P. Wherry, Omaha, presented a postmortem sinus drill.

Dr. William J. McNally, Montreal, made a Report on Research Work. No discussion.

The following Symposium was presented:

"The Histology of Inflammation of Nasal Mucous Membrane and the Rôle of the Reticulo-Endothelial System," Dr. Leroy A. Schall, Boston.

"Reformation of Mucous Membrane in Twenty Reoperative Cases of Chronic Maxillary Sinusitis," Dr. Gregor McGregor, Toronto, Canada.

"Reformation of Mucous Membrane," Dr. Charles D. Knowlton, Boston.

"An Experimental and Clinical Study of Histiocytes in Acute and Chronic Inflammation of the Accessory Sinuses," Dr. Ralph A. Fenton, Portland, Oregon.

"The Study of Nasal Mucous Membrane in Hanging Drop Tissue Culture," Dr. P. J. Ross, Boston.

"Summary," Dr. Harris P. Mosher, Boston.

Dr. Samuel R. Skillern, Philadelphia, read a paper entitled "The Place of Diathermy for Tonsillectomy." Discussed by Drs. Robert F. Ridpath, Philadelphia; James A. Flynn, Washington, D. C.; George L. Richards, Fall River, Mass.; E. M. Josephson, New York, and Samuel R. Skillern.

The Thursday afternoon session adjourned.

FRIDAY, SEPTEMBER 23, 2:00 P.M.

Section on Ophthalmology

The Friday afternoon session convened at two o'clock, Dr. S. Hanford McKee presiding.

Presentation of New Instruments:

Dr. Harvey J. Howard, St. Louis, demonstrated a hospital bed designed especially for patients undergoing cataract operation or any operation on the eyes.

Dr. H. W. Grant presented, for Dr. Frank E. Burch, St. Paul, Minnesota, pressed celluloid shields for eye operations.

Dr. Edward Stieren, Pittsburgh, presented intraocular forceps.

Dr. W. D. Rowland, Boston, presented a new tenotomy hook and lid retractor.

Dr. Walter B. Lancaster, Boston, read a paper entitled "Astigmatic Charts Versus Crossed Cylinders: Results in One Hundred Office Cases." Discussed by Drs. Edward Jackson, Denver; S. Judd Beach, Portland, Maine, and Walter B. Lancaster.

Dr. Hugh S. McKeown, New York, read a paper entitled "Detachment of the Retina — the Guist Operation: Report of Cases." Discussed by Drs. Harry S. Gradle, Chicago; Daniel B. Kirby, New York; Peter C. Kronfeld, Chicago, and Hugh S. McKeown.

Dr. Jonas S. Friedenwald, Boston, read a paper entitled "Monocular Myopia." Discussed by Drs. Meyer Wiener, St. Louis; E. M. Josephson, New York, and Jonas S. Friedenwald.

Dr. Conrad Berens, in conjunction with Dr. Olga Sitchevska, New York, presented a paper entitled "Complete Discission of the Crystalline Lens." Discussed by Drs. E. C. Ellett, Memphis; Meyer Wiener, St. Louis; V. M. Hicks, Raleigh, N. C.; Geo. Slocum, Ann Arbor, Michigan, and Conrad Berens.

Dr. W. D. Rowland, Boston, read a paper entitled "Visual Field Studies: Pseudo Upper Form Field Contractions." Discussed by Dr. John Evans, Brooklyn, N. Y.

The Thirty-Seventh Annual Meeting of the American Academy of Ophthalmology and Otolaryngology adjourned *sine die*.

THURSDAY, SEPTEMBER 22, 7:30 P.M.

Business Meeting

The annual Business Meeting of the Academy convened at seven-thirty Thursday evening, the President, Dr. S. Hanford McKee, in the chair.

The following reports were read:

REPORT OF SECRETARY-TREASURER

This report summarizes the financial and membership situation as of September 1, 1932.

Finance

During the year 1931 there was received from:		
Dues and applications	$18,180.70	
Transaction Sales	152.50	
Convention items	8,197.20	
Interest on capital investments	4,883.18	
Profit on Bonds sold	1,215.87	$32,629.45
Against which there was disbursed:		
Research	$ 5,500.00	
Transactions	2,872.24	
Honoraria		
Comptroller	100.00	
Secretary-Treasurer	400.00	
Meetings and Conventions	9,636.85	
Administration	7,253.10	
Accrued Interest	86.60	25,851.79
Balance		$ 6,777.66

Leaving a credit balance of $6,777.66, which was passed to the Reserve Fund. Not counting research appropriations, this gives an operating cost of $10.55 per member.

SUMMARY OF ASSETS, JANUARY 1, 1932

Cash on hand	$ 2,100.27	
Capital investments (book value)	98,201.72	$100,301.99

The market value of the capital assets over the book value on January first showed a shrinkage of 18.5 per cent, which seemed very good, since in the average of trust holdings over the United States a shrinkage of 37 per cent has been reported.

In July the Citizens Building Company of Cleveland was reorganized and we received bonds in the new company to the amount of $5,000.00, and common stock of no par value. This amount had been previously put in the profit and loss account in the audit of 1929. Dr. Large deserves credit for this reorganization.

These accounts have been audited by a certified public accountant and a copy of his report is herewith attached.

Partial report for the eight months of 1932 — January 1st to September 1st:

So far in 1932 there has been received:

Dues and applications	$17,994.98	
Transaction sales	388.85	
Convention items	1,428.71	
Interest on capital investments	2,915.00	$22,727.54

Against which there has been disbursed:

Research	$ 2,700.00	
Transactions	4,076.31	
Honoraria:		
Comptroller	100.00	
Secretary-Treasurer	400.00	
Trust Company	106.00	
Meetings and Conventions	2,037.60	
Administration	3,491.98	
Accrued Interest	29.22	$12,941.11

Leaving a net credit balance of	$ 9,786.43
Bonds purchased for capital account	$ 7,577.50

Gift of 5 shares Ophthalmic Publishing Company stock — par value of $100.00 — donated by Dr. Mary Heard, sister of Dr. Heard.

Resources as of September 1, 1932

Cash on hand	$ 4,310.62	
Trust holdings	105,879.22	$110,189.84

MEMBERSHIP SUMMARY

In good standing, September 1, 1931		1,791
Elected at French Lick Springs		138
Total, January 1, 1932		1,929

The following members have resigned:

Dr. Fred F. Agnew, Independence, Iowa
Dr. Virginius Dabney, Washington, D. C.
Dr. Carl F. Felt, Philadelphia, Pennsylvania
Dr. W. G. Harrison, Birmingham, Alabama
Dr. B. D. LaForce, Altadena, California
Dr. McCluney Radcliffe, Philadelphia, Pennsylvania
Dr. John F. Rowland, Hot Springs National Park, Arkansas
Dr. W. H. Thomasson, Newport, Kentucky
Dr. J. E. Cogan, Cleveland, Ohio
Dr. C. E. Miller, Muncie, Indiana

Total membership		1,929
Lost by death and resignation	36	1,895
Candidates pending		130
Total carried on books as of September 1, 1932		2,023

As Executive Secretary, representing your operating officers, I desire to express appreciation for your splendid cooperation thus far, and in behalf of those soon to be elected for the coming year I ask the same cooperation.

W. P. WHERRY, M.D.
Executive Secretary-Treasurer

Moved, by Dr. H. P. Mosher, that this report be accepted as read. Motion seconded by Dr. Joseph C. Beck, and carried.

In the absence of Dr. H. W. Lyman, the necrologist, the names of deceased members were read by Dr. W. P. Wherry, the members standing in silence for one minute in memory of the departed.

NECROLOGY REPORT

The Academy has lost the following members by death during the past year:

Dr. F. W. Alter, Toledo, Ohio
Dr. Garrett Guy Ash, Bradford, Pennsylvania
Dr. William Ellery Briggs, Sacramento, California
Dr. Louis D. Brose, Evansville, Indiana
Dr. Margaret F. Butler, Philadelphia, Pennsylvania
Dr. A. E. Bulson, Fort Wayne, Indiana
Dr. Edward Fayette Cary, New York, New York
Dr. George Strong Derby, Boston, Massachusetts
Dr. Calvin Ross Elwood, Menominee, Michigan
Dr. Joseph Brown Farrior, Tampa, Florida
Dr. John Farquahar Fulton, St. Paul, Minnesota
Dr. Frederic John Haas, Leavenworth, Kansas
Dr. Corydon Ford Heard, Erie, Pennsylvania
Dr. Norman MacLeod Heggie, Jacksonville, Florida
Dr. Mike Clement Jenkins, Pratt, Kansas
Dr. Jefferson D. Kiser, Bartlesville, Oklahoma
Dr. Charles William Kollock, Charleston, South Carolina
Dr. Joseph Albert Lassalle, Montreal, Quebec, Canada
Dr. George Street McReynolds, Sr., Temple, Texas
Dr. Leland Osgood Mauldin, Greenville, South Carolina
Dr. Emil Mayer, New York, New York
Dr. Lloyd Leslie Nelson, Norfolk, Nebraska
Dr. Theodore B. Schneideman, Philadelphia, Pennsylvania
Dr. David William Stevenson, Akron, Ohio
Dr. Lea Wadsworth Tindolph, Olean, New York
Dr. Frank J. Van Kirk, Bellingham, Washington
Dr. John Otis Weaver, Shenandoah, Iowa

Respectfully submitted,

H. W. LYMAN, M.D., *Necrologist*

Dr. Francis William Alter

Dr. Francis William Alter, of Toledo, Ohio, was born in that city on June 21, 1869.

His early education was obtained in a parochial school and the Toledo High School. He subsequently attended a preparatory school in New York City. On taking up the study of medicine, he attended, for one term, the Toledo Medical College, then matriculated in the University of New York (Bellevue Hospital Medical College), from which he received his M.D. degree in 1896. After a year's service as an interne in the New Amsterdam Hospital, of New York City, he took a postgraduate course in the New York Ophthalmic and Aural Institute, and then returned to Toledo, where he practiced for five years.

On the expiration of that period, he went abroad, and, for eighteen months, studied under physicians in Vienna, Austria. In 1904, Dr. Alter resumed his professional activities in Toledo, and has since devoted his attention to the treatment of diseases of the eye, ear, nose and throat. For twelve years he was connected with St. Vincent's Hospital, and later was a member of the staff of Mercy Hospital. In 1924, he opened the first eye, ear, nose and throat hospital in Lucas County, Ohio, and the institution is now conducted as a day hospital, and used exclusively for this branch of professional service.

For eight years, Dr. Alter was one of the trustees of the Toledo Academy of Medicine, of which he was elected president in 1924; and for thirty years was one of its officers, representing the Academy in every capacity except that of treasurer. He was ex-secretary of the Lucas County, Ohio, Medical Society; a member of the American Medical Association, the American Academy of Ophthalmology and Otolaryngology, and the American College of Surgeons, in which he held a fellowship for fifteen years.

Dr. Alter died during August, 1932, leaving his widow and eight children surviving him.

Dr. Garrett Guy Ash

Dr. Garrett Guy Ash, of Bradford, Pennsylvania, received his medical education at Western Pennsylvania Medical College Pittsburgh, graduating in 1907.

He was on the staff of the Bradford Hospital at the time of his death.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

Dr. Ash died October 14, 1931, at the age of forty-nine years.

Dr. William Ellery Briggs

Dr. William Ellery Briggs was born at Sharon, Ohio, March 31, 1853. When eight years of age, his family moved to Wadsworth, Ohio, where he attended the public schools, and later graduated from the University of Akron. In 1877, he graduated from the Medical Department of the University of Wooster.

After his graduation, Dr. Briggs went to London and Vienna for postgraduate work in diseases of the eye, ear, nose and throat. In 1879, he located in Sacramento, where he practiced until the date of his death.

He was a member of the Sacramento Society for Medical Improvement, The California Medical Association, The Northern District Medical Society, The American Medical Association, The Pacific Coast Oto-Ophthalmological Society, The American Academy of Ophthalmology and Otolaryngology, The International Ophthalmology Association, and a fellow in the American College of Surgeons. He was the founder and president of the Northern California Eye, Ear, Nose and Throat Society.

Dr. Briggs died November 5, 1931, at Sacramento, California, leaving surviving him his widow and two children.

Dr. Louis D. Brose

Dr. Louis D. Brose, of Evansville, Indiana, received his medical education at the University of Pennsylvania School of Medicine, Philadelphia, graduating in 1881.

He was demonstrator and professor of anatomy, Evansville Medical College, until it dissolved in 1885; and, for many years, oculist and aurist to St. Mary's Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and the American College of Sur-

geons; past president of the Vanberburgh County Medical Society; delegate to the Pan-American Medical Congress in Washington, D. C., in 1893, the International Medical Congress in Berlin in 1900, and the International Otological Congress in Boston in 1912.

Dr. Brose died November 15, 1931, at the age of seventy-two years.

Dr. Margaret F. Butler

Dr. Margaret F. Butler, of Philadelphia, Pennsylvania, received her medical education at the Woman's Medical College of Pennsylvania, Philadelphia, graduating in 1894.

She was professor of laryngology, rhinology, and otology at her alma mater, and rhinologist and otolaryngologist at the Woman's Hospital of Philadelphia at the time of her death.

She was a member of the American Academy of Ophthalmology and Otolaryngology and the American College of Surgeons.

Dr. Butler died October 16, 1931, at the age of seventy years.

Dr. Albert Eugene Bulson

Dr. Albert Eugene Bulson was born in Chicago, December 16, 1867.

He was graduated from the Michigan State College in 1888, and from the Rush Medical College, in Chicago, in 1891. He took postgraduate work in Philadelphia, New York, and Europe. He had practiced in Fort Wayne, Indiana, since 1892, being a specialist in ophthalmology and otolaryngology.

He was professor of ophthalmology and otolaryngology at the Fort Wayne College of Medicine from 1894 to 1908. He was professor of Ophthalmology at Indiana University School of Medicine, Indianapolis, and Bloomington, since 1908, and had been head of that department of the university since 1928. For many years he served on the surgical staffs of St. Joseph's and the Methodist Hospitals at Fort Wayne.

He was a member of the Fort Wayne Medical Society, the American Academy of Ophthalmology and Otolaryngology, the Indiana State Medical Association, and the Amedican Medical Society.

He was editor of the Fort Wayne Medical Journal from 1894 to 1908; collaborator in the Encyclopedia of Ophthalmology; and, in addition, was author of many scientific articles dealing with diseases of the eye, ear, nose and throat.

He was past president of both the Fort Wayne Medical Society and the Northern Tri-State Medical Association; secretary and chairman of the Section of Ophthalmology of the American Medical Association from 1903 to 1911; and was vice-speaker of the House of Delegates of the American Medical Association since 1929.

Dr. Bulson was also treasurer of the Knapp Testimonial Fund for Research in Ophthalmology, and had been since its establishment in 1910; and served on the American Board for Ophthalmic Examinations from 1921 to 1924, representing the American Medical Association.

Dr. Bulson died July 17, 1932, at Fort Wayne, Indiana, leaving his widow and two children surviving him.

Dr. Edward Fayette Cary

Dr. Edward Fayette Cary, of New York City, received his medical education at the Baltimore Medical College, graduating from that college in 1906.

At the time of his death he was on the staff of the Manhattan Eye, Ear, and Throat Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

Dr. Cary died on August 14, 1932, at the age of fifty-five years.

Dr. George Strong Derby

Dr. George Strong Derby was born in Boston, Massachusetts, May 29, 1875.

After his graduation from the Harvard Medical School, Dr. Derby went abroad to carry on his studies in ophthalmology under Fuchs, Snelling and Axenfeld, returning to this country in 1903. He then joined the staff of the Massachusetts Eye and Ear Infirmary, and of the Carney Hospital.

In 1924, he was appointed professor of ophthalmology at Harvard University.

He was a member of the American Academy of Ophthalmology and Otolaryngology; the American Ophthalmological Society; The American Board for Ophthalmic Examinations; The New England Ophthalmological Society; and The American College of Surgeons. At one time he was secretary of the Section of Ophthalmology of the American Medical Association, and later chairman of that section. In 1916, he received the Knapp medal of the Section of Ophthalmology of the American Medical Association.

During the World War he served with the Harvard Unit and the English Ophthalmic Center in Boulogne, finally being discharged, at the end of the war, with the rank of Lieutenant-Colonel.

He organized the Infants' Hospital in Boston, and established a wet nursery. He was deeply interested in the National Association for the Prevention of Blindness, and, at the time of his death was president of the Suffolk Medical Society.

Dr. Derby died in Boston, Massachusetts, on December 12, 1931.

Dr. Calvin Ross Elwood

Dr. Calvin Ross Elwood was born in Waterford Center, Michigan, June 24, 1870. He graduated at Pontiac High School in 1889. In 1890, he entered the University of Michigan and received his M.D. degree in 1894.

After graduation, he was assistant physician at Eastern Michigan Asylum for one year.

During 1895 and 1896 he studied with Dr. Fleming Carroll of Ann Arbor. In July, 1896, he located in Menominee, Michigan, where he engaged in the practice of medicine, devoting his whole time to diseases of the eye, ear, nose and throat. In 1909, he spent several months in Vienna, studying.

During the World War, he joined the Medical Corps as first lieutenant, and was made major, June 10, 1919.

Dr. Elwood died on September 18, 1931, leaving his widow and two children surviving him.

Dr. Joseph Brown Farrior

Dr. Joseph Brown Farrior was born in Campbellton, Florida, April 14, 1881. His parents moved to Chipley, Florida, when he was young, where he spent his boyhood. He attended college at the Southern University in Greensboro, Alabama, and later entered Tulane Medical University, graduating in 1905.

He was in general practice several years. In 1911, he completed a postgraduate course at the University of Chicago, specializing in eye, ear, nose and throat.

He opened on office in Tampa, Florida, in June, 1912, his practice being limited to the eye, ear, nose and throat. He was active in civic work and took a leading part in the campaign which led to the construction of the present Tampa Municipal Hospital.

In 1930 he was president of the Hillsborough County Medical Society. He was a member of the Florida Medical Association, the Southern Medical Association, American Medical Association, the Seaboard Railway Surgeons' Association, and the American Academy of Ophthalmology and Otolaryngology.

Dr. Farrior died in Tampa, Florida, October 3, 1931, leaving his widow and six children surviving him.

Dr. John F. Fulton

Dr. John F. Fulton was born in York, Pennsylvania.

He was educated in the University of Pennsylvania, where he received not only his medical degree, but his master's degree in the arts and the degree of doctor of philosophy.

He practiced two year in Altoona, Pennsylvania, and then came to St. Paul, Minnesota, in 1882. He was on the original faculty of St. Paul Medical College, founded in the early eighties, and about ten years later absorbed by the University of Minnesota Medical School. Upon this merger, he joined the university faculty, and, at the time of his death was emeritus professor of ophthalmology.

He was also on the staffs of St. Luke's and Miller Hospitals, and, for many years, was oculist on the surgical staff of the Northern Pacific, Great Northern, and other northwest railroads.

He was a past president of the Ramsey County and Minnesota Academy of Medicine, of which he was one of the founders, and the Minnesota Academy of Ophthalmology.

He was secretary, Section on Ophthalmology, 1885-1886; secretary, Section on Laryngology and Otology, 1893-1894, and chairman, 1894-1895; trustee, American Medical Association, 1901-1904; member of the American Academy of Ophthalmology and Otolaryngology; and fellow of the American College of Surgeons.

Dr. Fulton died in St. Paul, February 1, 1932.

Dr. Frederic J. Haas

Dr. Frederic J. Haas was born in Leavenworth, Kansas, June 14, 1876. He received his early schooling in Leavenworth, both grade and high school, and attended and graduated from the University Medical College, Kansas City, Missouri, in 1907. After graduation he left for Vienna, Austria, and Berlin, for postgraduate work in 1908 and 1909.

Dr. Haas was consultant oculist to the National Military Home of Kansas. For a number of years, and until his death, he was executive officer, St. John's Hospital Medical Staff, and a member of Cushing Memorial Hospital Medical Staff. He was also lecturer on eye, ear, nose and throat to the Nurses' Training Schools, St. John's Hospital and Cushing Hospital.

He was a member and censor of Leavenworth County Medical Society; a member of the Kansas State Medical Society; a fellow in the American Medical Association; a member of the American Medical Association of Vienna; and a member of the American Academy of Ophthalmology and Otolaryngology.

Dr. Haas died on June 6, 1932.

Dr. Corydon Ford Heard

Dr. Corydon Ford Heard was born in Ripley, New York, November 9, 1876.

Dr. Heard was graduated from the Medical College of the University of Michigan in 1898. He was assistant in the Department of Eye, Ear, Nose and Throat in 1898-9, and then returned to Northeast, Pennsylvania, where he was associated

with his father and brother in general practice, laying a firm foundation for his special work.

In 1908, and again in 1911, he studied abroad, and, on his return in 1912, he located in Erie, Pennsylvania, limiting his practice to otolaryngology. He still remained consultant at St. Mary's College, in Northeast, Pennsylvania, where, since its location there in 1880, father and sons had been physicians in charge.

He was also on the staffs of Hamot and St. Vincent's Hospitals, and consultant at St. Joseph's Orphanage, and the Zem Zem Crippled Children's Home.

He was a fellow of the American Medical Association, and a member of the American Academy of Ophthalmology and Otolaryngology and state and local societies.

Dr. Heard died in Erie, Pennsylvania, December 17, 1931.

Dr. Norman MacLeod Heggie

Dr. Norman MacLeod Heggie, of Jacksonville, Florida, was born in Brampton, Ontario, Canada, October 14, 1876.

He received his early education in the schools of Canada, and was graduated from the University of Maryland in 1902.

For two years he was interne in the Presbyterian Eye and Ear Hospital, after which he studied extensively in Scotland and Germany. He came to Jacksonville, Florida, in March, 1906.

He was a past president of the Duval County Medical Society, president of St. Luke's Hospital Staff, president of Riverside Hospital Staff, and chief of the eye staff of St. Vincent's Hospital. He was a past governor of the American College of Surgeons, a member of the American Academy of Ophthalmology and Otolaryngology, American Board of Ophthalmology, American Medical Association, Southern Medical Association, Southeastern Surgical Congress, Southern Railway Surgeon's Association, Florida State Medical Association, and Duval County Medical Society.

During the World War, Dr. Heggie was commissioned a major in the Medical Corps of the United States Army.

Dr. Heggie died May 9, 1932, leaving surviving him his widow and two children.

Dr. Mike Clement Jenkins

Dr. Mike Clemont Jenkins was born near Pratt, Kansas, October 10, 1886.

He received his early education in Pratt, and his medical degree from the Kansas City Medical College in 1911.

Following his graduation, he practiced a short time in Manhattan, Kansas, before establishing a practice in Pratt, eighteen years ago. Only a few years before his death, he had completed a course in his specialty of eye, ear, nose and throat diseases at Harvard University.

He was a member of the American Academy of Ophthalmology and Otolaryngology; and a member of the State Board of Medical Registration and Examination at the time of his death. He was also on the staff of the Ninnescah Hospital.

Dr. Jenkins was killed November 13, 1931. He is survived by his widow and four children.

Dr. Jefferson D. Kiser

Dr. Jefferson D. Kiser, of Bartlesville, Oklahoma, received his medical education at the Kentucky School of Medicine, Louisville, Kentucky, graduating from that school in 1894.

He was on the staff of the Washington County Memorial Hospital, at Bartlesville, Oklahoma, at the time of his death.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

He died April 14, 1932, at the age of sixty-three years.

Dr. Charles Wilson Kollock

Dr. Charles Wilson Kollock, of Charleston, South Carolina, received his medical education at the University of Pennsylvania School of Medicine, in Philadelphia, graduating from that college in 1881.

Dr. Kollock was emeritus professor of rhinology and laryngology of the Medical College of the State of South Carolina; and laryngologist and rhinologist to the Roper Hospital at Charleston, South Carolina.

Dr. Kollock served in the Medical Department during the World War.

He was a member of the South Carolina Medical Association, American Academy of Ophthalmology and Otolaryngology, and the American College of Surgeons.

Dr. Kollock died on September 23, 1931, at the age of seventy-four years.

Dr. Joseph Albert Lassalle

Dr. Joseph Albert Lassalle graduated from the School of Medicine and Surgery of Montreal in 1895.

He served in the Ophthalmological Clinic of his alma mater, becoming chief of clinic after the death of Dr. Chrétien Zavagg. Later he was appointed professor of ophthalmology in the University of Montreal.

He was a member of the staff of L'Hôtel Dieu, having served that institution for thirty-five years.

He was a member of The French Society of Ophthalmology, The French Society of Otolaryngology, The Society of Laryngology of the Hospitals of Paris, The Society of Ophthalmology of Montreal (of which he was president for two years), The American Academy of Ophthalmology and Otolaryngology. He was also a fellow of the American College of Surgeons.

The last honor bestowed upon him was his election to the presidency of the Medical Society of Montreal. By a tragic coincidence his funeral occurred on the day he was to have presided at his first meeting.

He died at his home, January 16, 1932, at the age of fifty-nine years.

Dr. George Street McReynolds, Sr.

Dr. George Street McReynolds, Sr., was born in Christian County, Kentucky, February 11, 1872. He was educated in the public schools of Hopkinsville, Kentucky; attended the South Kentucky College there; Tulane University at New Orleans, Louisiana; and was graduated from the College of Physicians and Surgeons at Baltimore, Maryland, in 1898.

He interned in the Baltimore City Hospital and the Presbyterian Eye, Ear, Nose and Throat Hospital.

Dr. McReynolds practiced for two years at Memphis, Tennessee, and, in August, 1902, moved to Temple, Texas, where,

for many years, he was associated with the King's Daughters' Hospital and Clinic.

He was a member of the Bell County Medical Society; Texas State Society; American Medical Association; a fellow of the American College of Surgeons; the American Academy of Ophthalmology and Otolaryngology; the Texas State Ophthalmological and Otolaryngological Society; The American Bronchoscopic Society; a past councillor of the Texas State Association; and past president of the Bell County Medical Society.

Dr. McReynolds demonstrated the removal of foreign bodies from the air passages of dogs, over the entire state, and contributed a number of articles to the literature of bronchoscopy.

Dr. McReynolds is survived by his widow and a brother, Dr. John O. McReynolds, of Dallas, Texas. Dr. McReynolds' death occurred suddenly at Clovis, New Mexico, August 26, 1931.

Dr. Leland Osgood Mauldin

Dr. Leland Osgood Mauldin was born at Pickens, South Carolina, on May 10, 1878.

He received his early education in the common and high schools near and at Pickens Court House and at Clemson College, where he graduated with a B.S. degree. He was graduated from the South Carolina Medical College in 1903.

Dr. Mauldin engaged in general practice in Pickens, South Carolina, for a short time, later receiving an appointment as medical examiner for the Bureau of Pensions, Washington, D. C. While in Washington, Dr. Mauldin took postgraduate work in the study of diseases of the eye, ear, nose and throat. After remaining in Washington for one year, he went to London, England, where he took further postgraduate work in the Royal London Ophthalmic Hospital and the London Central Ear and Throat Hospital, later going to the Allgeemeines Krankenhaus — a branch of the University of Vienna — for the same purpose.

In 1906, he established himself in Greenville, South Carolina, where he confined his practice to diseases of the eye, ear, nose and throat.

He was a member of the South Carolina Medical Association; American Medical Association; the Tri-State Medical Association of Virginia and the two Carolinas; the Southern Med-

ical Association; the American Academy of Ophthalmology and Otolaryngology; and a fellow in the American College of Surgeons.

He served as councillor for the Fourth District of the State Medical Association; as president of Greenville County Medical Society; and of the Staff of Greenville City Hospital. He also served as president of the South Carolina Medical Association for one year.

During the World War he was chairman of the Medical Advisory Board of First District of South Carolina.

He died at his home in Greenville, South Carolina, May 6, 1932, leaving his widow and three children surviving him. In his will he left all his surgical instruments and diagnostic equipment, as well as his medical library, to Greenville City Hospital.

Dr. Emil Mayer

Dr. Emil Mayer, distinguished laryngologist, died in New York on October 20, 1931.

In the fifty-four years of his active practice, he was an indefatigable worker and was prominently associated with many laryngological societies and institutions. Graduating in 1870 from the College of the City of New York, he turned first to pharmacy. Following his graduation in medicine in 1877, he became assistant-surgeon in the New York Eye and Ear Infirmary, and surgeon in the throat department of that institution, thirteen years later. He was, at various times, chief of clinic, attending laryngologist, and consulting laryngologist at Mt. Sinai Hospital, lecturer in the New York Polyclinic, and expert examiner City Civil Service Committee in the City of New York.

He was the American representative to the Fraenkel Birthday Celebration in 1906, when he was made an honorary member of the Berlin Laryngological Association.

He contributed frequently to the editorial pages of numerous journals, and held the position of abstract editor of the American Laryngological Association for several years.

He was a member of the Section on Laryngology of the American Medical Association, the New York Academy of Medicine, and the American Academy of Ophthalmology and Otolaryngology, of which he was president in 1921.

In 1922, Dr. Mayer was elected president of the American Laryngological Association; and, in 1927, became an honorary fellow of the American Laryngological, Rhinological and Otological Society.

In the last ten years of his life he interested himself in the study of local anesthetics, and collected extensive data upon the fatalities from these drugs. He was chairman of the Permanent Committee on Local Anesthesia of the Therapeutic Research Committee of the American Medical Association from 1924 until the time of his death. He contributed an extensive survey of the subject to the Loeb Memorial number of the Annals of Otology, Rhinology and Laryngology, which appeared in March, 1928.

Dr. Lloyd Leslie Nelson

Dr. Lloyd Leslie Nelson was born at Woodbine, Iowa, in 1886, and graduated from the Medical Department of Creighton University, at Omaha, Nebraska, in 1910.

The first six years of his practice were at Oakdale, Nebraska, doing general work.

He spent one year at the Manhattan Eye and Ear Hospital at New York City, taking up eye, ear, nose and throat work; and, for the past twelve years, devoted his practice to diseases of the eye, ear, nose and throat at Norfolk, Nebraska.

Dr. Nelson never married.

He died March 1, 1932.

Dr. Theodore B. Schneideman

Dr. Theodore B. Schneideman was born in Butler, Pennsylvania, November 9, 1861. He prepared for college at Rugby Academy, Philadelphia; graduated from Princeton University in 1881; and from Jefferson Medical College in 1883. He served for a year as interne in the Philadelphia General Hospital; engaged in the practice of general medicine for five years; and then began the practice of ophthalmology.

He was instructor in diseases of the eye in the Philadelphia Polyclinic and College for Graduates in Medicine, then adjunct-professor, and finally professor of ophthalmology in the same institution, which chair he held for over twenty years.

He was formerly oculist to St. Christopher's Hospital for Children, consulting ophthalmologist to the Woman's Hospital, and assistant-surgeon to Will's Eye Hospital.

For a number of years he was a collaborator of the Ophthalmic Year Book. He was editor of the transactions of the Philadelphia County Medical Society, and its secretary for seven years.

He was a member of the American Ophthalmological Society, The American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons.

In 1908, he married Dr. Florence Mayo, who was associated with him in practice.

Dr. Schneideman died in the Lankenau Hospital, October 23, 1931.

Dr. David William Stevenson

Dr. David William Stevenson, of Akron, Ohio, received his medical education at Rush Medical College, Chicago, graduating in 1890.

He was on the staffs of the Akron City and People's Hospitals in Akron.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and a fellow of the American College of Surgeons.

He died March 29, 1932, at the age of sixty-six years.

Dr. Lea Wadsworth Tindolph

Dr. Lea Wadsworth Tindolph, of Olean, New York, received his education at the University of Virginia Department of Medicine, Charlottesville, graduating in 1906.

He was on the staff of the Olean General Hospital at the time of his death.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

Dr. Tindolph died on April 25, 1932, at the age of fifty-nine years.

Dr. Frank J. Van Kirk

Dr. Frank J. Van Kirk was born June 14, 1867, at Hastings, Minnesota. He spent his childhood near Ripon, Minnesota, living on a homestead and growing wheat. From the profits of his efforts on the farm, he was enabled to pay his way through the University of North Dakota, being one of the first graduates of that institution.

On completion of his university course he entered the Railway Mail Service on the Great Northern Railroad, serving several years as railway postoffice clerk.

He later entered Hamlin University, graduating in medicine. After graduating from the latter institution, he acted as surgeon for the Great Northern Railway, attached to the Cascade Division during the building of the Cascade tunnel.

Later, he specialized in diseases of the eye, ear, nose and throat, studying under New York specialists and coming to Bellingham in 1900.

He was a member of the American College of Surgeons; Pacific Ophthalmological Society; American Medical Association; Washington State Medical Association; and Whatcom County Medical Association.

Dr. Van Kirk is survived by his widow and one brother, A. N. Van Kirk, of Minnesota.

Dr. John Otis Weaver

Dr. John Otis Weaver was born May 18, 1887, at Milford, Iowa. Coon Rapids was the family home for many years.

He largely earned his way through the University at Iowa City. His health broke down while he was in school, but, overcoming this handicap, he was graduated from the College of Medicine at the University of Iowa in the Class of 1914, and spent his interneship at the University Hospital.

His postgraduate work was taken in Chicago, and, during the first two years following his graduation, he practiced in Battle Creek, Iowa, with Dr. C. A. Hartley. Dr. Weaver came to Shenandoah, Iowa, in the summer of 1916.

During 1918 he was commissioned and placed in command of Evacuation Ambulance Unit No. 70, and took this unit to France. Later, in France, he was transferred as commanding officer of Evacuation Ambulance Unit No. 68, attached to the VI Army Corps.

Dr. Weaver died December 13, 1931, leaving surviving him his widow and two children.

REPORT OF AMERICAN BOARD FOR OPHTHALMIC EXAMINATIONS

The Board, to date, has conducted 39 examinations in various parts of the country, the first one being held in Memphis, December 13th and 14th, 1916; and the last at Montreal, September 19th, 1932.

Since the last meeting of the Academy at French Lick Springs, September 14th, 1931, there have been held the following examinations:

Boston, Mass.	December 21, 1931	15 candidates
New Orleans, La.	May 9, 1932	16 candidates
Montreal, Canada	September 19, 1932	28 candidates

Total number of certifications to date, 1,000

A supplement to the 1931 Directory was issued on January 1, 1932, and distributed gratis to all holders of the certificates as well as to the 139 libraries and medical schools to whom the Directory was sent without charge.

The Board wishes it to be known that all candidates for its certificate are now required to show that they have been engaged in the special study of and training in ophthalmology for one year, and have supplemented this by two years of clinical work in a special ophthalmic hospital or dispensary and with private practice, making altogether three years of preparation before they are eligible to take the examination.

S. J. Beach
F. Phinizy Calhoun

Moved by Dr. H. P. Mosher that this report be received and placed on file. Motion seconded and carried.

REPORT OF AMERICAN BOARD FOR OTOLARYNGOLOGY

DR. W. P. WHERRY: In the absence of Dr. Carmody I will make this report.

The American Board for Otolaryngology has functioned as usual. In New Orleans 60 candidates were examined, and recently in this city, 74. Up to September 1, 1932, the Board has certificated 1,962 candidates.

The Board has just completed a new loose-leaf Directory, the object being to maintain a complete directory each year. Supplements as well as replacement sheets will be furnished to those purchasing this directory without extra cost. There has been added a register of residencies available in the United States, with full information as to fees, if any. We have added to the directory advice on special courses for postgraduate work.

The Board feels very grateful to those who have subscribed for this directory.

On motion, duly seconded, this report was accepted and placed on file.

Reports of Standing Committees

AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

The report here submitted, like those previously given, can only be a report of progress; without the possibility of pointing to definite, specific results. The former recommendation of the Committee to use the designation of visual acuity in terms of the metric system is still very much neglected. The reverence for Snellen's suggestion of recognizing 20/20 as the expression for standard or "normal" vision still hinders a good understanding of the facts on the part of non-medical men. It should be understood that 10 meters is really a better approximation to parallelism of rays and of visual axes than 20 feet; and that the wide use of metric notation throughout the world makes adherence of English feet a peculiar custom, for which there is no justification, except the reluctance to make a change.

The effort to attain standardization and uniformity of test-cards is now an effort to get away from the unequal visibility

and wide variations in test letters. This is being carried on by a series of tests made on the eyes of young persons by competent scientific observers in departments of certain universities. There has been as yet no tabulation of results on which to base a useful report. It is probable that wide cooperation with scientific organizations giving attention to the matter would help in the attainment of results and purposes which are held in common; and as practical assistance can be secured in that way the committee proposes to seek such cooperation. The cooperation of the Bureau of Standards has already been promised. Papers and discussions on points of visual physiology and optics before local or national societies will help to a better understanding of subjects in which we are most interested.

The Committee has examined the following instruments:

The simplified Zeiss lensometer was approved for simplicity.

The new cheiroscope (American Optical Company) is a modification of Maddox's apparatus, and is of value in training amblyopic eyes.

The Keystone Service consists of:

1. The Telebinocular — a new, improved stereoscope for office and home use.
2. The correct Eyescope — a Holmes model stereoscope with wooden cabinet. This serves as a base for drawing exercises and for use with split or standard slides; designed for home use, simple in operation and reasonable in price.
3. "O" series of charts.
   (a) 15 pairs of split slides for providing prism exercise. The prism effect depends upon the amount of separation of the two pictures.
   (b) 70 standard size stereographs.
      I. "F" series — seemingly duplicate slides of the same scene, but one is with, and the other is without stereoscopic effect; used in diagnosing and developing stereopsis.
      II. A-B-C-D-E series of slides, some easy and some difficult to fuse. Because of the minute detail and depth, many are well-suited for the development of accommodation.

4. P. G. series of charts consisting of vision, suppression, vergence, and deduction charts.
5. Twelve color fusion plates for developing simultaneous binocular vision, especially in marked amblyopia.
6. Correct eyegraphs for cheiroscopic exercises for right or left eye, for developing amblyopic eyes.
7. The book of stereoscopic technic which fully describes each slide and its use, both from a training and diagnostic standpoint.
8. Home training set consisting of a correct eyescope, a number of charts from each group, and a booklet giving detailed instructions for their use. The ophthalmologist may check which group of exercises is indicated in the individual case.

Respectfully submitted,

EDWARD JACKSON, *Chairman*
S. JUDD BEACH
CHARLES A. BAHN
WILLIAM L. BENEDICT
E. V. L. BROWN
ALFRED COWAN
CONRAD BERENS, *Secretary*

Moved by Dr. Burt R. Shurly that this report be accepted. Motion seconded by Dr. H. P. Mosher, and carried.

COMMITTEE TO INVESTIGATE THE TREATMENT OF INCIPIENT CATARACT

Unfortunately, the chairman of your Committee has been called home. He has asked me to make a report of progress for the Committee. It has been examining papers and data which have come to its attention, but sufficient time has not elapsed to fully analyze and correlate them. One of the members of your Committee has been conducting research along these lines. Your Committee begs to be continued.

Respectfully submitted,

DANIEL B. KIRBY

(This report was accepted).

EXTRAMURAL GRADUATE INSTRUCTION

The Extramural Graduate Instruction Committee of the Academy was appointed in 1930 for the purpose of investigating ways and means of organizing postgraduate instruction in our specialty.

Its activity has been confined to the need of the practicing physician who is recognized as a specialist in diseases of the ears, nose and throat, who desires a short, intensive course to enable him to refresh his basic work and obtain knowledge concerning the new, worth while developments in the field.

After a thorough discussion of this project at several of the Council Meetings, and also with a number of the leaders of the Academy, the committee gained the impression that there was both need and desire for such courses. Furthermore, the committee felt that with some stimulation and preparation the members of the Academy were capable of preparing courses that might, in comparison, be more beneficial than the courses given by foreign specialists brought to this country for the same purpose. Accordingly, a questionnaire was sent to Academy members who have had teaching experience, asking them to signify their willingness to prepare a thorough course; to select their own topics; and to specify the time of the year that would be most convenient for them to present such a course. One hundred and twenty-nine questionnaires were submitted to ophthalmologists, with seventy-one favorable replies. One hundred and twenty-seven questionnaires were sent to otolaryngologists, again with seventy-one favorable replies. In the two groups, there were thirty-three refusals, and eighty-one failed to reply. A copy of the same letter and questionnaire was sent to the deans of all medical schools.

The committee advises that the Academy should assume no financial obligation in sponsoring these courses. But with the information derived from the questionnaire, and from experience with the instructional courses given in connection with the annual meeting of the Academy, it is felt that the Academy is in a position to act as booking agent for the universities, or medical societies, who desire to present such courses. It recommends that a standard fee be charged for such courses, and that the instructors be given an honorarium, with all expenses paid.

Very enthusiastic letters have been received concerning the proposed courses. A few comments were unfavorable, but in every case, these were due entirely to a misunderstanding, or to a lack of information; and when the aims of the committee were explained adequately, the unfavorable statements were retracted in each instance.

The committee reports that there is sufficient material and information available so that assistance can be rendered whenever and wherever such courses are desired.

THE COMMITTEE:
W. V. MULLIN, M.D., *Chairman*
HARRY S. GRADLE, M.D.
W. L. BENEDICT, M.D.

PREVENTION AND AMELIORATION OF DEAFNESS

During the past year your Committee has undergone a process of reorganization to enlarge the scope of its activities and make possible better work. It is impossible to report in definite form the results of all your Committee's efforts. They have borne fruit in many ways which are not directly tangible.

All the members, through addresses before many medical, educational and lay organizations and through personal contacts, have sought to create a better understanding of the factors constituting the problem of deafness prevention and the care of the hard of hearing and the deaf.

A very definite and satisfactory results of efforts among the organizations of the hard of hearing has been to warn them against allowing their members to fall into the hands of the quack, or to waste their substance on fake cures or useless and possibly harmful mechanical devices sold to restore the hearing.

Partly due to the work of your Committee there has been a gratifying increase in the adoption of the periodic audiometric hearing test for school children. Not less than two hundred cities are now using this method with great satisfaction — a gain of one hundred per cent in two years.

A notable advance in the teaching in schools for the deaf which should be known to every otologist is found in the recent employment of instruments giving high degrees of vacuum tube amplification whereby many children with heretofore unrecog-

nized residual hearing are now taught correct speech through the ear. The early adoption of this improved method of instruction should be recommended by the otologist.

This Committee is engaged in formulating plans for bringing about better relations between the manufacturer and vendors of electric hearing aids to the end that there shall be a higher standard of ethical cooperation in rendering service to our hard of hearing patients.

In the belief that there is a large field for further endeavor along the lines of our activities, your Committee is now working on a more definitely organized plan for increased effort.

With the approval of the President, we would enlarge the Committee by the appointment of Dr. Gordon Berry of Worcester, Massachusetts; Dr. Thomas E. Carmody of Denver, Colorado, and Dr. Edward F. Ziegelman of San Francisco, California, as additional members.

Respectfully submitted,

HORACE NEWHART, *Chairman*
JOHN F. BARNHILL
E. H. CAREY
HOWARD V. DUTROW
A. A. HAYDEN
FREDERICK T. HILL
GEORGE L. TOBEY
CLAUDE T. UREN

PUBLICITY AND LEGISLATION

Your Committee on Publicity and Legislation has notified the special journals of the Montreal session.

We are indebted to the local press for the excellent reports of our deliberations. The representatives have been courteous and most efficient and your Committee takes this opportunity to express to each our appreciation of their cooperation, tact and skill.

We have further corresponded with the Licensing Board of every state in the union in relation to regulations applying to those who wish to practice a specialty, and many of our correspondents have expressed a desire to assist in the establishment of legal standards. As one of the participating bodies in the

formation of the American Board for Ophthalmic Examinations, and the National Board of Otolaryngology, your legislative committee feels that the Academy should take an early and prominent part in the establishment of legal standards. New Jersey has recently proposed a plan for accrediting specialists and controlling specialism by the State Medical Society. Your Committee, therefore, recommends that the Academy go on record as approving legislation directed toward the better protection of the public by legal regulation of the use of the term "specialist."

A few years ago your present Committee on Legislation and Publicity proposed that we enter into an agreement with a journal devoted to ophthalmology, and one devoted to otolaryngology, whereby the papers read in the scientific sessions be published and news of the organization be placed before the members. When that proposal was placed before this Academy it was accepted. Your Committee respectfully calls your attention to the necessity of making some such arrangement and trusts that appropriate steps will be taken to consummate the plan.

Respectfully submitted,

ARTHUR J. BEDELL, *Chairman*
JOHN J. SHEA

Moved by Dr. Edward Jackson that this report be received and adopted. Motion seconded and carried.

TUNING FORKS AND TESTS OF HEARING

Last year your Committee reported on a complete series of tuning forks made by B. E. Eisenhour, of which the medium pitched ones are overtone-free. There was also a discussion of a rather comprehensive series of sounding rods made of the same metal as the forks. This metal, you will recall, is not only rust-proof and tarnish-proof, but is very light in weight and much less expensive, so that the cost of construction of forks and rods is less than for the best imported forks. For many years the unweighted A (108 double vibrations) fork of Bezold has been the one most commonly used by otologists in the Weber and Schwabach test. This is a most excellent fork and is practically overtone-free, is very heavy and has a long period of vibration. It is at the same time a very expensive instrument, particularly since the duty on foreign instruments was so greatly

increased in the last tariff act. Mr. Eisenhour has now succeeded in duplicating this fork in the new magnesium aluminum alloy of which the other forks of this series are made. This new fork is a splendid one, very light in weight, and vibrates a long time. It is true that during the first few seconds of its vibration a rather loud overtone is heard, but this can be entirely eliminated by placing around the ends of the prongs a rather wide rubber band, as was originally suggested by Bezold himself.

This Committee recommends this fork most highly and believes it will give excellent service to the otologic profession. The Committee recommends the adoption of this report and the continuance of the Committee for further study.

Respectfully submitted,

ROBERT SONNENSCHEIN, *Chairman*
E. G. GILL
L. W. DEAN

Moved by Dr. Joseph C. Beck that this report be accepted. Motion seconded and carried.

NATIONAL OTOLARYNGOLOGICAL LIBRARY

DR. JOSEPH C. BECK: This is the first, and it might be said to be a preliminary report. We have just organized as a committee, with Dr. Burt R. Shurly as chairman, and the following members from the different national societies — The American Laryngological, The American Otological, The Triological, The Pacific Coast, and the Sections in the American Medical Association:

L. W. DEAN
JOHN F. BARNHILL
T. E. CARMODY
RALPH A. FENTON
JOSEPH C. BECK

The Committee has not had time to do very much work, but they are prepared to go to work, and ask for a continuance of this committee.

Moved by Dr. Robert Sonnenschein that the report be adopted. Motion seconded and carried.

REPORT OF COMMITTEE ON NOMENCLATURE OF DISEASE

To remedy the existing confusion and lack of authority in Medical Nomenclature in the United States, an informal conference was held in New York, March 22nd, 1928. At this meeting the present organization, *The National Conference on Nomenclature of Disease*, was formed, the primary object of which was to unite the important national societies representing medicine, surgery, and their specialties, the federal medical services and interested national health organizations and life insurance interests in an effort to develop a standard *National Nomenclature of Disease*. A representative executive committee was charged with the responsibility for the necessary research and for the preparation of a basic plan.

At the meeting in November, 1930, the basic plan was officially adopted. It provides for a dual method of classification — etiological and topographical. Each disease entity is conceived of as having a site — usually in an organ or part of an organ, or it may be in the body as a whole. The site is the anatomical or topographical part of the designation. Secondly, each disease is conceived of as having a cause. This constitutes the second part of the designation. Thus the eye is subdivided into some fifty anatomical parts. The etiological categories are, most of them, common to all parts of the body and are used for each of the anatomical systems. There are a dozen major categories, each subdivided into about ten, and each of these into ten more when necessary. The actual working out of the plan cannot be readily understood without having the lists in hand for study.

A comprehensive detailed classification of disease has now been prepared in accordance with the basic plan which should meet the needs of the largest and most highly-specialized hospitals. There is also an abridged version designed for smaller institutions. A special hospital chiefly concerned with one branch of medicine would use the detailed list for its specialty, and the abridged list, perhaps, for recording diseases outside its special field. The two versions are printed in the same volume. There is an index, containing not only the preferred names, but as far as possible *all* names in current use, with cross references to the preferred titles. Existing nomenclatures have been consulted and their best features freely borrowed. The purpose is not to

introduce new or different names, but to select as standard what seem to be the best of those in current use. It is hoped that by degrees many of the synonyms will drop out of use and the standard become generally adopted. Factors favoring its getting into general use will be its adoption by publications, its use by teachers, and its dissemination through internes going out from hospitals which have adopted it.

It is important that the nomenclature be kept constantly abreast of the progress of medicine, and that it be subjected to periodical revision. Provision will be made for this, the cost being covered by the sale of the official edition of the Standard National Nomenclature of Disease.

The numerical system or code system has been adopted by many large hospitals, health departments, insurance companies, and federal departments for tabulating statistics and for other purposes. Accordingly the Standard Nomenclature has been adapted to this system; but the use of the numbers is entirely optional. The code numbers are not likely to be used in most private offices, but will be used in large hospitals, and so forth. Only those who have used such a system appreciate its possibilities and advantages.

The lists which have been prepared through the cooperation of the various sub-committees, with the invaluable guidance of the secretary, Dr. H. B. Logie, were formally adopted last December; and a limited edition will be published for distribution to a number of institutions where it will be used for about a year, and then revised and published for public sale to the general medical profession. If any of the ophthalmic hospitals or departments wish to give it a trial, the ophthalmic committee can probably arrange for the secretary, Dr. Logie, or someone like him who understands fully the whole plan, to visit the hospital and assist in getting it in operation.

There is active international interest in the subject, and there is reason to believe that international cooperation will be obtained. The widespread use of an authoritative and carefully-planned nomenclature cannot help but be instrumental in eliminating the use of many faulty and incomplete designations of disease. It is, therefore, hoped that it will exercise an influence upon the accuracy and precision of medical thought.*

* Much of this report is abridged from the reports of the President, Chairman of the Executive Committee, and Secretary of the larger committee.

The list of diseases of the eye prepared by your Committee is far from perfect. The task has proved more difficult than was expected. It is only fair to the rest of the Committee to say that the defects and errors in the list are due to the chairman, who was obliged in many cases to make decisions without the aid of the other members. The work has proved extremely interesting, and has convinced us of the importance of a *standard* and if possible an improved nomenclature. We believe that this represents the first step, at least, in that direction.

WALTER B. LANCASTER, *Chairman*
T. B. HOLLOWAY
J. M. WHEELER
CONRAD BERENS
SANFORD R. GIFFORD
JONAS S. FRIEDENWALD

Moved by Dr. S. J. Kopetzky that the report be adopted. Motion seconded and carried.

REPORT OF SENIOR MEMBER OF COUNCIL

The Council has accepted with regret the resignation of the Editors of the Transactions.

The Council desires to express its appreciation to Dr. Heard for the gift of five shares of capital stock — Americal Journal of Ophthalmology.

Because of the financial stringency the Council voted unanimously to carry an extra year members whose dues are already delinquent three years, except Dr. Ira A. Tripp, of Cleveland, who specifically requests his name be removed from the roll.

The question of issuing the Transactions in a quarterly form as contrasted to the present annual volume was discussed, and it was decided to make no change for the current year.

The proposal to finance the distribution of copies of the Directory — American Board of Otolaryngology — to the libraries of approved medical schools was approved.

Research appropriations for 1933 will be as follows:

Dr. William J. McNally	$1,500.00
Dr. Ralph A. Fenton	500.00
Dr. S. R. Gifford	500.00

Dr. C. S. O'Brien	500.00
Army Medical Museum	500.00
Trachoma	250.00
Standard Nomenclature of Disease	125.00

HARRIS P. MOSHER

Moved by Dr. W. V. Mullin that this report be adopted. Motion seconded and carried.

THE PRESIDENT: You will notice that the Constitution is printed in the back of the program, with the notation that "Due to various changes made from time to time in the Constitution the Judicial Committee recommended at the 1931 meeting that the complete Constitution, as it now stands, be published in the official program and that action on same be a matter of business this year." I would now like somebody to move the adoption of the Constitution as it appears in the present program.

DR. HARRIS P. MOSHER: I so move, Mr. President. (Motion seconded by Dr. W. V. Mullin, and carried).

Election of Members

DR. W. P. WHERRY: The Council has selected for election to honorary membership the following fellows:

Dr. William C. Bane ..... Denver, Colorado
Dr. H. M. Becker ..... Sunbury, Pennsylvania
Dr. S. V. Dabney ..... Louisville, Kentucky
Dr. Frank L. Dennis ..... San Diego, California
Dr. O. B. Dunn ..... Ironton, Ohio
Dr. D. D. McHenry ..... Oklahoma City, Oklahoma
Dr. H. Moulton ..... Fort Smith, Arkansas
Dr. T. E. Oertel ..... Augusta, Georgia
Dr. W. Scott Renner ..... Buffalo, New York

I move that the action of Council in the selection of these fellows for honorary membership be approved. (Motion seconded by Dr. J. F. Barnhill, and carried).

DR. W. P. WHERRY: The following candidates have been elected to fellowship in the Academy:

Andrew, James H., 163 Hancock St., Brooklyn, N. Y., Op.
Armstrong, E. Leslie, 205 W. 2nd St., Duluth, Minn., ALR

Athens, Alvin Glenwood, 425 Bradley Bldg., Duluth, Minn., Op.
Atwood, Edward A., 360 Park Ave., Paterson, N. J., ALR
Barr, Albert Stewart, First National Bank Bldg., Ann Arbor, Mich., Op.
Barton, William C., 104½ W. San Francisco St., Santa Fe, N. M., Op.
Bergmann, Milton Blaine, 959 Bushwick Ave., Brooklyn, N. Y., Op.
Bloom, C. Hugh, 65 North Third St., Easton, Pa., ALR
Boyes, Truman Leo, 100 West 59th St., New York, N. Y., Op.
Breeding, Earle Griffith, 1801 Eye St., N.W., Washington, D. C., ALR
Breuning, Paul Harold, 30 West 59th St., New York, N. Y., ALR
Brewster, Hyder Ford, Charity Hospital, New Orleans, La., ALR
Brobeck, Von Haller, 301 Ferguson Bldg., Colorado Springs, Colo., Op.
Buckman, Lewis Taylor, 83 South Franklin St., Wilkes-Barre, Pa., ALR
Bullard, Carleton William, 1825 Spruce St., Philadelphia, Pa., ALR
Cady, Frederick J., 507 Peoples Bldg. and Loan Bldg., Saginaw, Mich., ALR
Casten, Virgil Glenn, 520 Commonwealth Ave., Boston, Mass., Op.
Chainski, Edward L., 25 E. Washington St., Chicago, Ill., ALR
Cline, Samuel, 353 Commonwealth Ave., Boston, Mass., ALR
Cohen, William, 126 New York Ave., Brooklyn, N. Y., Op.
Connole, Joseph Vincent, 14 East Market St., Wilkes-Barre, Pa., Op.
Coombs, Arthur Jethro, 307 North Michigan Ave., Chicago, Ill., ALR
Cooper, Roy C., 510 South Aikin Ave., Pittsburgh, Pa., ALR
Culler, Arthur Merl, 530 Fidelity Medical Bldg., Dayton, O., Op.
Culpepper, William S., Station Hospital, Fort Munroe, Va., ALR
Darrough, Lawrence Earl, 4105 Live Oak St., Dallas, Tex., ALR
Davies, Morgan C., 269 E. State St., Columbus, Ohio, Op.
Dewar, Murray McColl, 609 Medical Arts Bldg., Grand Rapids, Mich., Op.
Dowling, Harvey Elwood, 1812 Eaton Tower, Detroit, Mich., Op.
Evans, Maurice George, 311 Commonwealth Ave., Boston, Mass., ALR

Evans, Sidney Saurin, 502 Physicians and Surgeons Bldg., Memphis, Tenn., ALR

Eves, Curtis Clyde, 1910 Spruce St., Philadelphia, Pa., ALR

Fine, Abraham, 1505 Ocean Ave., Brooklyn, N. Y., Op.

Freedman, Abraham O., 1396 St. Catherine St., W., Montreal, Canada, ALR

Friesen, J. Frank, 1208 Roosevelt Bldg., Los Angeles, Calif., ALR

Garrett, John Dempsey, 57 Stokes Bldg., Indianapolis, Ind., ALR

Goldcamp, Edward C., 810 Dollar Bank Bldg., Youngstown, O., ALR

Grimes, Charles Henry, 3546 N. Broad St., Philadelphia, Pa., ALR

Halper, Philip Allen, 30 North Michigan Ave., Chicago, Ill., OALR

Hirst, Otto C., 1930 Chestnut St., Philadelphia, Pa., ALR

Hoffman, William A., 231 W. Washington St., Chicago, Ill., Op.

Holloway, Joseph Minor, N. W. cor. Hanover and Charles, Fredericksburg, Va., ALR

Holzberger, John Conrad, 7005 Freshpond St., Brooklyn, N. Y., ALR

Huber, Walter A., 1114 Medical Arts Bldg., Tulsa, Okla., Op.

Jesberg, Simon, 500 South Lucas Ave., Los Angeles, Calif., ALR

Johnson, David Morrison, 327 East State St., Columbus, Ohio, ALR

Johnson, Henrietta May, 9th and H Sts., Broken Bow, Neb., Op.

Johnston, Kenneth Burns, 1509 Sherbrooke St., W., Montreal, Canada, Op.

Kammerling, Theodore S., 231 W. Washington St., Chicago, Ill., Op.

Kearny, Richard Augustine, The Woodward Apt., Washington, D. C., ALR

Kend, Lee, 400 West End Ave., New York, N. Y., ALR

Knight, John Swann, 1324 Professional Bldg., Kansas City, Mo., ALR

Koller, Louis Robert, Medical Arts Bldg., Minneapolis, Minn., ALR

Krimsky, Emanuel, 745 Eastern Parkway, Brooklyn, N. Y., Op.

Laird, Robert Glover, Metz Bldg., Grand Rapids, Mich., Op.

Lambert, Robert, 7 East 85th St., New York, N. Y., Op.

Lang, George Hermanne, 202 E. Liberty St., Savannah, Ga., ALR

Lupo, Carl Wilton, 578 Ovington Ave., Brooklyn, N. Y., ALR

Macnaughton, Benjamin Frank, 604 Medical Arts Bldg., Montreal, Canada, ALR

McAlester, Andrew Walker, 3rd, 2003 Bryant Bldg., Kansas City, Mo., Op.

McAskill, James Ernest, 500 Woolworth Bldg., Watertown, N. Y., ALR

McAuliffe, Gervais Ward, 110 West 55th St., New York, N. Y., ALR

McCollough, Thomas Boyle, 121 University Place, Pittsburgh, Pa., ALR

McDougall, J. Calhoun, 1001 Medical Arts Bldg., Atlanta, Ga., ALR

Marcotte, Raymond Henry, 215-A Main St., Nashua, N. H., ALR

Mendel, James Harold, Germantown Prof. Bldg., Philadelphia, Pa., ALR

Miller, Joseph Austin, 45 Johnson Ave., Newark, N. J., ALR

Moehle, Walter, 15 Schermerhorn St., Brooklyn, N. Y., Op.

Moll, Arthur Mackenzie, Powers Bldg., Grand Rapids, Mich., ALR

Mumma, Claude S., 163 E. Walton Place, Chicago, Ill., ALR

Neff, Eugene, 224 W. Washington Ave., Madison, Wis., Op.

Nippe, Leonard, 316 Michigan St., Toledo, Ohio, OALR

Ohly, John Henry, 22 Schermerhorn St., Brooklyn, N. Y., Op.

O'Rourke, Carroll, 208 Wayne Pharmacal Bldg., Fort Wayne, Ind., ALR

Orr, Eugene, Doctors Bldg., Nashville, Tenn., ALR

Otrich, Grover Cleveland, Commercial Bldg., Belleville, Ill., ALR

Patterson, Reese Williams, Medical Arts Bldg., Knoxville, Tenn., ALR

Payne, Brittain Ford, 1304 Walker Ave., Houston, Texas, Op.

Perry, Claude Severance, 40 South Third St., Columbus, O., Op.

Ray, Victor, Jr., 914 Carew Tower, Cincinnati, Ohio, Op.

Regan, James Joseph, 520 Commonwealth Ave., Boston, Mass., Op.

Sanders, Sam Houston, 899 Madison St., Memphis, Tenn., ALR

Sayad, William Yohannan, 1215 Harvey Bldg., West Palm Beach, Fla., ALR

Scheer, Henry Morris, 522 West End Ave., New York, N. Y., ALR

Schmidt, Otto V. M., 111 East 61st St., New York, N. Y., ALR

Schoenberg, Mark J., 1160 Park Ave., New York, N. Y., Op.

Shope, Edward Pierce Lentz, 511 Cooper St., Camden, N. J., Op.
Silva, Rafael, Departmento de Salubriad, Mexico City, Mexico, Op.
Simon, Emanuel, 72 Willett St., Albany, N. Y., ALR
Sitchevska, Olga L., 160 West 87th St., New York, N. Y., Op.
Slotkin, Herman Bennett, 1819 Pine St., Philadelphia, Pa., ALR
Stiles, Porter, Medical Arts Bldg., Birmingham, Ala., ALR
Stine, George Haeberle, 305 Burns Bldg., Colorado Springs, Colo., Op.
Straatsma, Clarence Ralph, 899 Park Ave., New York, N. Y., ALR
Sullivan, Robert Edward, Doctors Bldg., Nashville, Tenn., Op.
Swann, Cecil Collins, 101 Haywood St., Ashville, N. C., ALR
Tanner, James Wallace, 314 E. Grand Ave., Eau Claire, Wis., ALR
Terry, Theodore Lasater, 128 Newbury St., Boston, Mass., Op.
Townes, C. Dwight, 610 Heyburn Bldg., Louisville, Ky., Op.
Towson, Charles Emory, Germantown Prof. Bldg., Philadelphia, ALR
Tremble, George Edward, 1390 Sherbrook St., W., Montreal, Canada, ALR
Trotter, Homer A., 40 North St., Buffalo, N. Y., ALR
Vandevere, William Ewing, 810 Bassett Tower, El Paso, Texas, ALR
Walker, Clifford Black, 427 W. 5th St., Los Angeles, Calif., Op.
Walker, George Washington, 532 T. W. Patterson Bldg., Fresno, Calif., ALR
Walker, John Robert, 532 T. W. Patterson Bldg., Fresno, Calif., Op.
Weaver, Homer Virgil, 228 Cleveland Ave., N.W., Canton, Ohio, ALR
Weiss, J. Allan, 104 S. Michigan Ave., Chicago, Ill., ALR
Westervelt, H. O., 239 Pipestone St., Benton Harbor, Mich., ALR
Wilkerson, William Wesley, 1025 Bennie-Dillon Bldg., Nashville, Tenn., ALR
Woodruff, George Henry, 500 North Ottawa St., Joliet, Ill., ALR
Wright, Robert P., 1414 Drummond St., Montreal, Canada, ALR

DR. W. P. WHERRY: Mr. President, I move that this list of 112 candidates be elected to membership in the American Academy of Ophthalmology and Otolaryngology. (Motion seconded and carried.)

Election of Officers

DR. H. P. MOSHER: Mr. Chairman and Members of the Academy: I have the honor to report, for your Council, the following names for officers of this Academy for 1933:

President
DR. BURT R. SHURLY ..... Detroit

President-Elect
DR. JOHN M. WHEELER ..... New York

First Vice-President
DR. NELSON M. BLACK ..... Miami

Second Vice-President
DR. J. T. ROGERS ..... Montreal

Third Vice-President
DR. L. A. SCHALL ..... Boston

Comptroller
DR. S. H. LARGE ..... Cleveland

Executive Secretary and Treasurer
DR. W. P. WHERRY ..... Omaha

Secretary for Ophthalmology
DR. W. L. BENEDICT ..... Rochester

Secretary for Otolaryngology
DR. J. L. MYERS ..... Kansas City

Secretary for Instruction (Ophthalmology)
DR. HARRY S. GRADLE ..... Chicago

Secretary for Instruction (Otolaryngology)
DR. W. V. MULLIN ..... Cleveland

Member of Council
DR. E. H. CAREY ..... Dallas

Member Ophthalmological Board
DR. S. J. BEACH ..... Portland, Maine

Members Otolaryngological Board
DR. T. E. CARMODY ..... Denver
DR. W. P. WHERRY ..... Omaha

Editorial Board
SECRETARIAL BOARD

Member Committee on Physiological Optics
DR. JOHN EVANS ..... Brooklyn

Moved by Dr. Robert Sonnenschein that the secretary cast the ballot of the Academy for the candidates named.

DR. W. P. WHERRY: I hereby cast the unanimous ballot of those present for the officers named in the report of the senior member of the Council for the year 1933.

Selection of 1933 Convention City

DR. H. P. MOSHER: Mr. Chairman and Members of the Academy: Your Council recommends that the meeting place for 1933 be the city of Boston.

DR. BURT R. SHURLY: I move the adoption of this recommendation. (Motion seconded and carried.)

DR. H. P. MOSHER: As a resident of that benighted city, I would like to ask you to consider it carefully and begin to make your plans to complete your education by coming to Boston in 1933.

THE PRESIDENT: Is there anything further to come before this meeting? If not, I declare it adjourned.

ALPHABETICAL LIST OF MEMBERS

* Attended 1932 meeting. Number in parentheses indicates year in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Executive Secretary, Dr. W. P. Wherry, Medical Arts Bldg., Omaha, Nebr.

HONORARY FELLOWS

*BECK, JOSEPH C. ('01).....185 N. Wabash Ave., Chicago, Ill., ALR
*BIRKETT, H. S.....1190 Mountain St., Montreal, Canada, ALR
DUNDAS-GRANT, SIR JAMES.....148 Harley St., W. 1, London, Eng.
ELLIOT, LT.-COL. R. H.
54 Welbeck St., Cavendish Sq., W., London, Eng.
ELSCHNIG, PROF. A....Ferdinandstrasse 10, Prague, Czecho-Slovakia
GRAVES, BASIL
11 Devonshire Place, Upper Wimpole St., W. 1, London, Eng.
GRAY, ALBERT A.....Glasgow, Scotland
GRÓSZ, PROF. EMIL DE.....Budapest, VIII, Maria, V. 39, Op.
*JACKSON, EDWARD ('02).....314 Fourteenth St., Denver, Colo., Op.
JOHNSON, LINDSAY, Britannia Bldg., West St., Durban, Natal, Africa
KIRKPATRICK, LT.-COL. HENRY, 54 Welbeck St., W. 1, London, Eng.
LAWFORD, J. B.....Sagamore, New Road, Ashtead, Surrey, Eng.
LISTER, SIR WILLIAM T....24 Devonshire Place, W. 1, London, Eng.
NAGAR, PROF. F.....Freiestrasse 20, Zurich, Switzerland
TILLEY, HERBERT....University College Hospital, London, Eng., Op.
VAN DER HOEVE, PROF. J.....Rijnsburgerweg, 6 A, Leiden, Holland

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LIFE MEMBERS

ALLEN, J. H.....Republic Bldg., Denver, Colo., ALR
BANE, WILLIAM C.....Republic Bldg., Denver, Colo., OALR
BECKER, H. M.....49 S. 4th St., Sunbury, Pa., OALR
BROWN, EDWARD J. ('11)
900 Nicollet Ave., Minneapolis, Minn., OALR
DABNEY, S. G.....Starks Bldg., Louisville, Ky., OALR
DAY, EWING W.....121 University Place, Pittsburgh, Pa., OALR
DENNIS, F. L. ('06).Ferguson Bldg., Colorado Springs, Colo., ALR
DIXON, W. E.....1413 Spurgeon, Santa Ana, Calif., OALR
DUNN, O. B.....4th and Railroad Sts., Ironton, Ohio, OALR
FOSTER, HAL ('94).1426 Professional Bldg., Kansas City, Mo., ALR
INGERSOLL, J. M.('08)..1700 S. Bayshore Lane, Miami, Fla., ALR

LOCHARD, L. B.....Metropolitan Bldg., Denver, Colo., OALR
McHENRY, D. D. ('12)
Medical Arts Bldg., Oklahoma City, Okla., OALR
MAIRE, LEWIS E. ('06)
16710 Jefferson Ave., East, Grosse Point, Mich., OALR
MOULTON, H. (1900)
Merchants Nat'l Bank Bldg., Ft. Smith, Ark., OALR
OERTEL, T. E.....638 Greene St., Augusta, Ga., OALR
OWEN, FRANK S.....Medical Arts Bldg., Omaha, Nebr., OALR
POSEY, WM. C.....Radnor, Pa., Op.
RENNER, W. S. (1900)....341 Linwood Ave., Buffalo, N. Y., ALR
SCALES, J. W. ('96).....204½ Main St., Pine Bluff, Ark., OALR
SELFRIDGE, G. L.....384 Post St., San Francisco, Calif., ALR
STEIN, O. J.....Palos Verdes Estates, Calif., ALR
STEWART, T. M.....901 Union Trust Bldg., Cincinnati, O., OALR
STILLSON, H. .....Seaboard Bldg., Seattle, Wash., OALR
VINSONHALER, F.('02)....Urquhart Bldg., Little Rock, Ark., OALR
WOOD, CASEY A.....7 W. Madison St., Chicago, Ill., Op.
WRIGHT, JOHN W.....137 E. State St., Columbus, Ohio, Op.
YOUNG, H. B.....204 Jefferson St., Burlington, Iowa, Op.A

FELLOWS

A

ABBOTT, W. J. ('19)..Carnegie Medical Bldg., Cleveland, O., OALR
ABRAHAMSON, IRA A.('27).605 Traction Bldg., Cincinnati, O., Op.
ADAMS, A. L. ('05)....Professional Bldg., Jacksonville, Ill., OALR
ADAMS, C. F. ('08).....34 West State St., Trenton, N. J., OALR
ADAMS, CHARLES JOSEPH ('28)
101 W. Sycamore St., Kokomo, Ind., OALR
ADAMS, D. S.....508 Hume-Mansur Bldg., Indianapolis, Ind., OALR
ADAMS, ELDRIDGE S. ('20)
Aeronautics Branch, Dept. of Commerce, Washington, D. C., ALR
ADAMS, FRANK M.('24).122 Waterman St., Providence, R. I., ALR
ADAMS, FREDERICK W. ('20)
Roosevelt Clinic, 1114 Boylston Ave., Seattle, Wash., OALR
ADKINS, GEO. ELI ('29)...Jackson Infirmary, Jackson, Miss., ALR
*ADLER, FRANCIS HEED ('29).313 S. 17th St., Philadelphia, Pa., Op.
*AEBLI, RUDOLF ('31)....30 East 40th St., New York, N. Y., Op.
AGAN, WM. B. ('30).....510 Third St., Brooklyn, N. Y., Op.
AINSLIE, GEORGE ('21).....Selling Bldg., Portland Ore., OALR
ALBRIGHT, GEO. C. ('31)...102 S. Clinton St., Iowa City, Ia., ALR
ALBRIGHT, LT.-COM. P. M.('22)
M. C., U. S. Navy, U. S. Naval Hosp., Annapolis, Md., OALR
ALDEN, ARTHUR M. ('22).....Frisco Bldg., St. Louis, Mo., ALR
ALDERDYCE, WM. W. ('08)....513 Madison Ave., Toledo, O., OALR

ALEXANDER, GEO. J.....1930 Chestnut St., Philadelphia, Pa., ALR
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IOWA CITY

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MOORE, PAUL McNARY, JR. .....University Hospital, ALR
O'BRIEN, C. S. .....University Hospital, Op.
RUTHERFORD, C. W. .....Univ. Hosp., Dept. Ophth., Op.

KEOKUK

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MASON CITY

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TRIPP, LEROY R. .....Security Bldg., OALR

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KANSAS

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CHADWICK, IRA B. ..... Terminal Bldg., Op.

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ROBERTSON, EDW. N. ..... New Caldwell Bldg., OALR

ELDORADO

SHULTS, E. J. G. ..... Haines Bldg., OALR

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HUTCHINSON

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ALLEN, G. HENRY ..... Mills Bldg., OALR
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MARKS, SAMUEL BLACKBURN ..... Security Trust Bldg., ALR
OFFUTT, W. N. ..... 406 Security Trust Bldg., OALR
YATES, EARL C. ..... 190 N. Upper St., ALR

LOUISVILLE

BASS, A. L. ..... Heyburn Bldg., OALR
DABNEY, S. G. ..... Starks Bldg., OALR
DEAN, WALTER ..... 300 Francis Bldg., OALR
DULANEY, OCTAVIUS ..... 811 Brown Bldg., ALR
HALL, G. C. ..... 705 Brown Bldg., OALR
PFINGST, A. O. ..... Heyburn Bldg., Op.
SHAFER, J. J. ..... Starks Bldg., OALR
TOWNES, C. DWIGHT ..... Heyburn Bldg., Op.
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RICHMOND

DUNN, MURISON ..... OALR

LOUISIANA

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MOSS, OLIN W. ..... Weber Bldg., ALR

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BLUM, HENRY N. ..... 1211 Maison Blanche Bldg., Op.
BREWSTER, H. F. ..... Union Indemnity Bldg., ALR
COLLIER, GEO. BENJAMIN ..... 1317 Whitney Bldg., ALR
FUCHS, VALENTINE HENRY ..... Chaille Bldg., ALR
HASPEL, M. DAVID ..... Maison Blanche Bldg., OALR

HUME, JOHN R. .....Maison Blanche Bldg., ALR
LANDFRIED, C. J. .....607 Baronne Bldg., ALR
LEJEUNE, F. E. .....632 Maison Blanche Bldg., ALR
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TAQUINO, GEORGE J. .....208 Audubon Bldg., ALR
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BEACH, S. JUDD .....704 Congress St., Op.
CUMMINGS, GEORGE O. .....47 Deering St., ALR
GILBERT, FRANK Y. .....148 Park St., OALR
GORDON, CHAS. H. .....46 Deering St., ALR
HASKELL, ALFRED W. .....142 High St., Op.
HOLT, E. E., JR. .....723 Congress St., Op.
O'SULLIVAN, T. J. .....10 Pine St., ALR
SPALDING, J. A. .....627 Congress St., Op. A

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CLINE, SAMUEL .....353 Commonwealth Ave., ALR
CORBETT, J. J. .....520 Beacon St., OALR
DUNPHY, ED. B. .....520 Commonwealth Ave., Op.
EVANS, MAURICE GEO. .....311 Commonwealth Ave., ALR
FREEDMAN, L. M. .....475 Commonwealth Ave., ALR
GREENWOOD, ALLEN .....82 Commonwealth Ave., Op.
HURLEY, E. D. .....419 Boylston St., Op.
INGLIS, HARRY J. .....43 Bay State Road, ALR
KLEINERT, MARGARET NOYES .....82 Commonwealth Ave., ALR
KNOWLTON, CHARLES DAVISON.....520 Commonwealth Ave., ALR
LANCASTER, W. B. .....520 Commonwealth Ave., Op.
LEWIS, J. P. .....543 Boylston St., ALR
LOWELL, W. H. .....82 Commonwealth Ave., Op.
LURIE, MOSES H. .....483 Beacon St., ALR
MOSHER, HARRIS P. .....828 Beacon St., ALR
POIRIER, GEORGE H. .....60 Bay State Road, ALR
PORTER, CHAS. T. .....520 Commonwealth Ave., ALR
QUINCY, J. E. .....270 Commonwealth Ave., ALR
REGAN, JAS J. .....520 Commonwealth Ave., Op.
RICE, G. B. .....99 Bay State Road, ALR
RICHARDS, L. G. .....319 Longwood Ave., ALR
ROWLAND, W. D. .....220 Clarendon St., Op.
SCHALL, LEROY A.....270 Commonwealth Ave., ALR
SKIRBALL, J. J. .....353 Commonwealth Ave., Op.
SMYTH, DUNCAN C. .....375 Commonwealth Ave., ALR
TERRY, THEODORE L. .....128 Newbury St., Op.
THOMPSON, PETER H. .....308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE .....9 Massachusetts Ave., Op.
TOBEY, GEORGE L., JR. .....270 Commonwealth Ave., ALR
WALKER, WALDO W. .....99 Bay State Road, ALR
WELLS, DAVID W. .....Hotel Westminster, Op.

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FALL RIVER

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FRAMINGHAM

TOMB, EVERETT H. .....129 Concord St., ALR

GARDNER

BONE, HERMAN D. .....19 Pleasant St., OALR

HOLYOKE

BURKE, JOHN R. .....207 Elm St., OALR
HUSSEY, E. J. .....245 Maple St., OALR

LAWRENCE

CONLON, F. A. .....301 Essex St., OALR

LOWELL

WIGGIN, WM. IRVING .....202 Central St., ALR

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NEW BEDFORD

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NORTHAMPTON

COLLINS, J. D. .....187 Main St., ALR
DOW, FRANK E. .....4 West St., Op.

PALMER

MOORE, G. A. .....353 Main St., OALR

PITTSFIELD

THOMPSON, G. H. .....74 North St., OALR

SALEM

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SPRINGFIELD

BYRNES, H. F. .....67 Chestnut St., OALR
CARLETON, RALPH .....10 Temple St., Op.
GALLAGHER, JOHN H. C. .....1537 Main St., ALR
IRWIN, VINCENT J., JR. .....1634 Main St., OALR
IRWIN, V. J., SR. .....1634 Main St. ,OALR

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BAKER, HARRY B. .....59 Main St., OALR

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BERRY, GORDON ..... 36 Pleasant St., ALR
CAHILL, JOHN WM. ..... 390 Main St., OALR
CROISSANT, CHAS. A. ..... 390 Main St., ALR
CROSS, A. E. ..... 390 Main St., Op.
ESTABROOK, CHAS. T. ..... 857 Pleasant St., OALR
GIBBY, HAROLD JAMES ..... 27 Elm St., ALR
HOLZER, WM. F. ..... 36 Pleasant St., Op.
MESSIER, A. EUGENE ..... 390 Main St., OALR
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SLOCUM, GEORGE ..... First Nat'l Bank Bldg., Op.

BATTLE CREEK

FRASER, R. H. ..... Old Merchants Tower, ALR
HAUGHEY, WILFRED H. ..... 303 Post Bldg., OALR
SLEIGHT, R. D. ..... 303 Post Bldg., Op.
WENCKE, CARL G. ..... Battle Creek Sanitarium, ALR

BAY CITY

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BEATTIE, ROBERT ..... David Whitney Bldg., OALR
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MCCLELLAND, CARL C. .....1857 David Whitney Bldg., OALR
MAIRE, LEWIS E. .....710 Kresge Bldg., OALR
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LILLIE, W. IVAN ..... Mayo Clinic, Op.
NEW, GORDON B. ..... Mayo Clinic, L
PRANGEN, AVERY DE H. ..... Mayo Clinic, Op.
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WAGENER, HENRY P. ..... Mayo Clinic, Op.

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NELSON, ALLAN LOUIS .....830 Lowry Medical Bldg., Op.
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WINONA

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WALKER, GEORGE H. .....172 Main St., OALR

WORTHINGTON

STANLEY, C. R. .....508 Tenth St., ALR

MISSISSIPPI

GREENVILLE

MONTGOMERY, D. CAMERON.....Leyser Bldg., ALR
PAYNE, VIRGIL LEROY .....Weinberg Bldg., ALR

GULFPORT

MCWILLIAMS, CHAS. A. .....G. & S. I. Bdlg., ALR

JACKSON

ADKINS, GEORGE E. .....Jackson Infirmary, ALR
HARRIS, WM. ROBIN .....Lamar Life Bldg., ALR

NATCHEZ

GAUDET, LUCIAN S. .....405 Franklin St., OALR

OXFORD

GUYTON, B. S. .....512 Van Buren, OALR

VICKSBURG

BELL, M. H. .....First National Bank Bldg., Op.
EDWARDS, C. J. .....First National Bank Bldg., OALR
HOWARD, E. F. .....First National Bank Bldg., ALR
JONES, EDLEY H. .....First National Bank Bldg., OALR

MISSOURI

CARTHAGE

POST, WINFRED B. .....304 Grant St., OALR
POWERS, EVERETT .....147 E. Third St., OALR

COLUMBIA

SNEED, CARL M. .....506 W. Broadway, OALR

EXCELSIOR SPRINGS

SHOEMAKER, FERDINAND .....U. S. Veterans' Hospital, OALR

KANSAS CITY

ALLEN, CHAS. E. ..... 712 Waldheim Bldg., OALR
ALTRINGER, A. N. ..... Professional Bldg., ALR
BEAL, HOMER A. ..... 1002 Argyle Bldg., ALR
BEIL, J. W. ..... Bryant Bldg., OALR
BELLOWS, GEORGE E. ..... 1500 Professional Bldg., Op.
BLACK, W. BYRON ..... Professional Bldg., ALR
BLAKESLEY, T. S. ..... Professional Bldg., OALR
CURDY, R. J. ..... 1102 Grand Ave., Op.
CURRAN, E. J. ..... 1805 Fed. Res. Bank Bldg., Op.
DAVIS, HARRY B. ..... Bryant Bldg., OALR
DIXON, OTTO J. ..... Professional Bldg., ALR
EUBANK, AMBROSE E. ..... 636 Argyle Bldg., OALR
FOSTER, HAL ..... 1426 Professional Bldg., ALR
GILLILAND, O. S. ..... Professional Bldg., ALR
HEDRICK, HAROLD B. ..... 910 Rialto Bldg., ALR
HOWARD, J. W. ..... Professional Bldg., OALR
KEITH, W. E. ..... Professional Bldg., OALR
KNIGHT, JOHN S. ..... Professional Bldg., ALR
LANING, J. HALCOMBE ..... 907 Rialto Bldg., ALR
LEMOINE, A. N. ..... Rialto Bldg., Op.
LICHTENBERG, J. S. ..... Rialto Bldg., Op.
LORIE, ALVIN J. ..... Commerce Bldg., ALR
LUX, PAUL ..... 409 Commerce Bldg., ALR
MCALESTER, ANDREW W., JR. ..... Bryant Bldg., Op.
MCALESTER, ANDREW W., 3RD ..... Bryant Bldg., Op.
MCCARTY, VIRGIL W. ..... Professional Bldg., OALR
MCKEE, JOSEPH W. ..... Professional Bldg., Op.
MILLER, HUGH ..... Chambers Bldg., OALR
MOTT, JOHN S. ..... 1302 S. Paseo Place, OALR
MYERS, JOHN L. ..... 815 Shukert Bldg., OALR
PAINTER, A. M. ..... Professional Bldg., ALR
ROBERTS, S. E. ..... Professional Bldg., ALR
SCHUTZ, W. H. ..... Bryant Bldg., OALR
SHUMATE, D. L. ..... 500 Argyle Bldg., OALR
SIMPSON, M. B. ..... Professional Bldg., ALR
THOMASON, H. E. ..... Rialto Bldg., ALR
TEALL, RAYMOND E. ..... Professional Bldg., ALR
YAZEL, HERMAN E. ..... 733 Rialto Bldg., OALR

ST. JOSEPH

FORGRAVE, L. ROBERT ..... 204 Logan Bldg., OALR
MINTON, WM. H. ..... Carby Bldg., OALR
PROUD, WILLARD C. ..... Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M. ..... Frisco Bldg., ALR
ALVIS, BENNETT Y. ..... 900 Carleton Bldg., Op.
ARBUCKLE, MILLARD F. ..... 1011 Beaumont Med. Bldg., ALR

BARDENHEIER, F. G. A. ..... Frisco Bldg., ALR
BEISBARTH, CARL C. ..... 3720 Washington Ave., Op.
BIRSNER, LOUIS J. ..... Missouri Bldg., ALR
BLACK, W. D. ..... Metropolitan Bldg., ALR
BOEMER, L. C. ..... 3720 Washington Blvd., ALR
BROWN, CLYDE O. ..... 3720 Washington Blvd., ALR
BURNS, S. S. ..... Missouri Theater Bldg., ALR
CALHOUN, JAMES GRAHAM ..... Metropolitan Bldg., Op.
CHARLES, JOS. W. ..... 3720 Washington Ave., Op.
CONE, ALFRED J. ..... McMillan Hospital, ALR
COSTEN, J. B. ..... Beaumont Medical Bldg., ALR
DEAN, L. W. ..... Medical School, Washington Univ., ALR
DOBBS, OTTO ROSS ..... 2435 N. Grand Blvd., ALR
DONNELL, N. R. ..... Metropolitan Bldg., Op.
DYER, C. P. ..... 408 Humboldt Bldg., Op.
EBER, CARL THEODORE ..... 1006 Carlton Bldg., Op.
EIMER, CHARLES EUGENE ..... 316 Lister Bldg., ALR
FERRIS, DAVID P. ..... 1000 Carleton Bldg., ALR
FINEBERG, MAXWELL ..... 3858 Westminster Place, ALR
FREIMUTH, L. E. ..... University Club Bldg., ALR
GLICK, HARRY N. ..... 3115 S. Grand Blvd., ALR
GOLDSTEIN, M. A. ..... 3858 Westminster Place, ALR
GREEN, JOHN ..... 3720 Washington Blvd., Op.
GREENE, M. L. ..... Missouri Theater Bldg., Op.
GUGGENHEIM, L. K. ..... 308 N. 6th St., ALR
GUNDELACH, C. A. ..... 825 University Club Bldg., ALR
HANSEL, FRENCH KELLER ..... Beaumont Medical Bldg., ALR
HARDESTY, J. F. ..... 706 Missouri Theater Bldg., Op.
HARDY, W. F. ..... Humboldt Bldg., Op.
HOBART, CARL ..... Metropolitan Bldg., Op.
HOOSS, C. H. A. ..... 3115 S. Grand Blvd., Op.
HOURN, GEORGE E. ..... 513 University Club Bldg., ALR
HOWARD, HARVEY J. ..... Park Plaza, Op.
JACOBS, MAX W. ..... Missouri Theater Bldg., Op.
JAMES, WM. M. ..... 640 S. Kingshighway, Op.
JONES, VINCENT L. ..... 3511 Washington Ave., Op.
KELLEY, ISAAC D., JR. ..... 3720 Washington Blvd., ALR
LAMB, HARVEY D. ..... 826 Metropolitan Bldg., Op.
LEAVY, C. A. ..... Metropolitan Bldg., ALR
LUEDDE, W. H. ..... Metropolitan Bldg., Op.
LYMAN, HARRY W. ..... Carleton Bldg., ALR
MCMAHON, BERNARD JOHN ..... Missouri Theater Bldg., ALR
MASON, ROY EARL ..... Frisco Bldg., Op.
MILLER, H. ED. ..... 728 Missouri Bldg., ALR
MYERS, EUGENE LEE ..... Wall Bldg., ALR
NEMOURS, PAUL ROLAND ..... 3852 Greer Ave., ALR
PAYNE, R. J. ..... University Club Bldg., ALR
PELZ, MORT D. ..... Missouri Bldg., ALR
PFINGSTEN, C. F. ..... 6370 Waterman Ave., ALR

POOS, GROVER H. ..... Univ. Club Bldg., OALR
POST, LAWRENCE T. ..... Metropolitan Bldg., Op.
POST, M. HAYWARD ..... Metropolitan Bldg., Op.
POTTER, ALLEN B. ..... 3725 Kingshighway, ALR
PROETZ, A. W. ..... Beaumont Bldg., ALR
REIM, HUGO ..... 417 Metropolitan Bldg., Op.
RYAN, L. M. ..... Wall Bldg., ALR
SAUER, W. E. ..... Beaumont Medical Bldg., ALR
SCHLUER, E. P. ..... Metropolitan Bldg., ALR
SCHOLZ, R. P. ..... Metropolitan Bldg., ALR
SCHWARTZ, FREDERICK O. ..... Metropolitan Bldg., Op.
SENSENEY, EUGENE T. ..... 316 Lister Bldg., ALR
SHAHAN, W. E. ..... Metropolitan Bldg., Op.
SHOEMAKER, W. A. ..... 1006 Carleton Bldg., Op.
SIMON, FREDERICK C. ..... 914 Arcade Bldg., ALR
SMIT, WM. M. ..... 719 Missouri Theater Bldg., ALR
TOOKER, C. W. ..... Carleton Bldg., Op.
WESTLAKE, SAMUEL B. ..... Beaumont Med. Bldg., ALR
WIENER, MEYER ..... 900 Carleton Bldg., Op.
WOOD, V. VISSCHER ..... Beaumont Medical Bldg., ALR
WOODRUFF, FREDERICK E. ..... Metropolitan Bldg., Op.

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TITSWORTH, GUY ..... 111 W. 4th St., OALR

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COFFELT, THEODORE A. ..... 1240 Benton Ave., OALR
KLINGNER, THOS. O. ..... 318 Landers Bldg., OALR
SMITH, CARLIE SOUTER ..... 214 N. Jefferson Ave., OALR

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MORSE, A. W. ..... 507 Phoenix Bldg., OALR
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BRIGGS, F. W. ..... OALR

ROUNDUP

LEWIS, GEORGE A. ..... Wall Bldg., OALR

NEBRASKA

BROKEN BOW

JOHNSON, HENRIETTA MAY ..... 9th and H Sts., Op.

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KOEBBE, EZRA EDWIN ..... Box 53, ALR

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FOX, CHAS. H. ..... 5 West 21st St., OALR

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BROOKS, EARL B. ..... 939 Stuart Bldg., OALR
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HOMPES, J. J. ..... Sharp Bldg., OALR
SANDERSON, DAVID D. ..... Stuart Bldg., OALR
THOMAS, JAMES W. ..... Sharp Bldg., OALR
ZEMER, S. G. ..... Stuart Bldg., OALR

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FAIRCHILD, NORA M. ..... Medical Arts Bldg., Op.
HANEY, WM. P. ..... 1500 Medical Arts Bldg., OALR
JUDD, J. H. ..... Medical Arts Bldg., Op.
KNODE, A. R. ..... 708 Medical Arts Bldg., ALR
KULLY, B. M. ..... Medical Arts Bldg., ALR
OWEN, F. S. ..... Medical Arts Bldg., OALR
POTTER, GEORGE B. ..... Medical Arts Bldg., OALR
POTTS, J. B. ..... Medical Arts Bldg., ALR
ROMONEK, PHILIP L. ..... Medical Arts Bldg., OALR
RUBENDALL, C. ..... Medical Arts Bldg., OALR
STOKES, WM. H. ..... Medical Arts Bldg., Op.
SWAB, CHARLES M. ..... Medical Arts Bldg., Op.
UREN, C. T. ..... 537 City National Bank Bldg., ALR
WHERRY, W. P. ..... 1500 Medical Arts Bldg., OALR

NEVADA

RENO

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UPHAM, HELEN F. ..... 305 Third Ave., OALR

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KLINE, ORAM R. ..... 414 Cooper St., ALR
SHOPE, ED. P. L. ..... 511 Cooper St., Op.

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HACKENSACK

GREENFIELD, WILLIAM JOHN ..... 50 Anderson St., ALR

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EAGLETON, WELLS P. ..... 15 Lombardy St., ALR
HAHN, WM. H. ..... 15 Lombardy St., Op.
HUGHES, L. W. ..... 965 Broad St., OALR
HURFF, J. WALLACE ..... 86 Washington St., OALR
KEIM, WILLIAM F. ..... 25 Roseville Ave,. ALR

MILLER, JOSEPH A. .....45 Johnson Ave., ALR
O'CONNOR, D. F. .....671 Broad St., OALR
ORTON, HENRY B. .....24 Commerce St., ALR
PEER, L. A. .....965 Broad St., ALR
QUINBY, W. O'G. .....14 James St., OALR
SHERMAN, ELBERT S. .....671 Broad St., Op.
WOOD, EARL LEROY .....192 Roseville Ave., ALR
ZEHNDER, A. CHARLES .....188 Roseville Ave., Op.

ORANGE

EMERSON, LINN .....Metropolitan Bldg., OALR

PATERSON

ATWOOD, ED. A. .....360 Park Ave., ALR

PLAINFIELD

CHILDERS, ROBERT JEFFERSON .....604 Park Ave., ALR
VAN HORN, A. F. .....514 Central Ave., ALR

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FORT BAYARD

GILL, ARTHUR E. .....U. S. Veterans' Hospital, OALR

SANTA FE

BARTON, WM. C. .....104½ W. San Francisco St., Op.

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DOWLING, J. IVIMEY .....116 Washington Ave., OALR
HINMAN, E. E. .....146 Washington Ave., ALR
JUDGE, HARRY V. .....46 Willett St., Op.
SIMON, EMANUEL .....72 Willett St., ALR
TEBBUTT, H. K. .....240 State St., ALR

BATAVIA

WILSON, ROBERT G. ..... Kresge Bldg., ALR

BINGHAMTON

KINNE, SANFORD HALSEY ..... 73 Main St., ALR
RANDALL, HIRAM ..... 81 Main St., ALR
WATSON, H. DE WITT ..... 151 Front St., OALR

BROOKLYN

AGAN, WM. B. ..... 510 Third St., Op.
ANDREW, JAS. H. ..... 163 Hancock St., Op.
BERGMANN, MILTON B. ..... 959 Bushwick Ave., Op.
BERNSTEIN, P. F. ..... 1265 President St., ALR
BUONAGURO, MICHAEL J. ..... 589 Lorimer St., Op.
COHEN, WM. ..... 126 New York Ave., Op.
CUTINO, R. M. ..... 165 Clinton St., Op.
DECOSTE, S. H. ..... 1179 Dean St., ALR
DE YOANNA, ALFRED A. ..... 111 Pierrepont St., ALR
EBELING, WM. B. ..... 54 Greene Ave., Op.
EVANS, J. N. ..... 23 Schermerhorn St., Op.
FEASTER, HENRY J. ..... 461 77th St., ALR
FINE, ABRAHAM ..... 1505 Ocean Ave., Op.
GOLDING, JOSEPH E. ..... 170 Hancock St., Op.
GREENFIELD, SAMUEL D. ..... 169 New York Ave., ALR
HANCOCK, J. C. ..... 135 Cambridge Pl., Op.
HARGITT, CHARLES A. ..... 17 Schermerhorn St., Op.
HAUPT, WM. C. ..... 1235 Dean St., ALR
HOLZBERGER, JOHN C. ..... 7005 Freshpond St., ALR
JAMESON, P. CHALMERS ..... 139 Montague St., Op.
KRIMSKY, EMANUEL ..... 754 Eastern Parkway, Op.
KRIMSKY, JOSEPH ..... 263 Eastern Parkway, OALR
KRUSKAL, I. D. ..... 863 Prospect Pl., Op.
LLOYD, RALPH I. ..... 14 Eighth Ave., Op.
LOMBARDO, MELCHIORE ..... 142 Joralemon St., Op.
LUPO, CARL W. ..... 578 Ovington Ave., ALR
MENGER, LOUIS CHARLES ..... 202 Weirfield St., ALR
MOEHLE, WALTER ..... 15 Schermerhorn St., Op.
O'BRIEN, G. R. ..... 307 Sterling Place, ALR
OHLY, JOHN H. ..... 22 Schermerhorn St., Op.
ROGERS, ROBERT M. ..... 680 St. Johns Pl., Op.
THOMASHEFSKY, M. ..... 135 Eastern Parkway, ALR
WEETH, CHAS. R. ..... 37 Remsen St., ALR

BUFFALO

ANDREWS, H. D. ..... 33 Allen St., Op.
BENNETT, ARTHUR G. ..... 150 N. Pearl St., Op.
BEYER, LOUIS J. ..... 449 Delaware Ave., ALR
BLAAUW, E. E. ..... 190 Ashland Ave., Op.
BOZER, HERRMANN EUGENE ..... 468 Delaware Ave., ALR

BROWN, C. M. .....510 Delaware Ave., ALR
COTT, C. C. .....104 Dorchester Road, ALR
COWPER, H. W. .....543 Franklin St., Op.
EDSON, R. A. .....957 Delaware Ave., Op.
FAIRBAIRN, JOHN F. .....925 Delaware Ave., ALR
FLAGG, J. D. .....158 Anderson Place, Op. A
GLOSSER, HERBERT H. .....448 Franklin St., Op.
HEALY, JOHN F. .....925 Delaware Ave., ALR
KOENIG, IVAN J. .....40 North St., Op.
LE WIN, THURBER .....112 Linwood Ave., Op.
LEWIS, F. PARK .....454 Franklin St., Op.
LUHR, A. F. .....40 W. North St., Op.
MCKEE, OTTO S. .....83 Bryant St., ALR
MARCH, CLARA A. .....465 Ashland Ave., Op.
PHILLIPS, WM. LINTON .....131 Linwood Ave., Op.
RENNER, W. S. .....341 Linwood Ave., ALR
TROTTER, HOMER A. .....40 North St., ALR
WEED, HARRY M. .....196 Linwood Ave., Op.
WURTZ, W. J. M. .....333 Linwood Ave., ALR

CORTLAND

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HIGGINS, R. P. .....20 Court St., OALR
WHITE, O. E. .....28 No. Main St., ALR

DUNKIRK

JACKLE, ARTHUR F. .....63 E. 4th St., Op.

FLUSHING

GOODE, EDWARD ALLEN .....143-28 Sanford Ave., ALR

GENEVA

SPENGLER, JOHN ARTHUR .....423 S. Main St., Op.

GLENS FALLS

BUCK, CLAIR A. .....Rogers Bldg., ALR
FIELDING, F. G. .....70 Ridge St., ALR

JAMAICA

BENDER, M. S. .....90-46 150th St., ALR

JAMESTOWN

REGER, H. S. .....Physicians Bldg., OALR

LOCKPORT

PARKER, HARRY L. .....88 East Ave., ALR
RINGUEBERG, EUGENE N. S. .....13 Main St., Op.

LONG ISLAND

SHERMAN, MAJ. A. ED. .....M. C., U. S. A., Mitchell Field, OALR

MIDDLETOWN

HICKS, WM. J. .....15 King St., ALR

MT. VERNON

KEEFE, WM. JOHN .....110 E. Sidney Ave., ALR
THOMSON, J. J. .....531 E. Lincoln Ave., ALR

NEW ROCHELLE

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HETRICK, L. E. .....260 North Ave., ALR
WOODRUFF, WALTER STUART.....421 Huguenot St., ALR

NEW YORK CITY

AEBLI, RUDOLF .....30 E. 40th St., Op.
ALMOUR, RALPH .....51 W. 73rd St., ALR
AUERBACH, JULIUS .....1016 Fifth Ave., ALR
BALLIN, M. J. .....20 E. 76th St., ALR
BASSEN, ED. J. .....698 Madison Ave., Op.
BEACH, B. S. .....140 W. 86th St., Op.
BERLINER, M. L. .....57 W. 57th St., Op.
BENDER, M. S. .....30 E. 40th St., ALR
BERENS, CONRAD .....35 E. 70th St., Op.
BOYES, T. L. .....100 W. 59th St., Op.
BRUCE, G. M. .....180 Fort Washington Ave., Op.
BREUNING, PAUL H. .....30 W. 59th St., ALR
BRUDER, JOSEPH .....57 W. 57th St., OALR
BULLWINKEL, H. GRIFFIN .....30 E. 76th St., ALR
CARTER, W. W. .....140 E. 54th St., ALR
CASTROVIEJO, RAMON .....30 W. 59th St., Op.
COHEN, MARTIN .....1 W. 85th St., Op.
COHN, FELIX .....945 West End Ave., ALR
COLBERT, ROBERT MARTIN .....57 W. 57th St., ALR
CORYA, HAROLD W. .....30 E. 40th St., ALR
COTT, D. N. .....25 Central Ave., St. George, Staten Isl., ALR
CUNNING, DANIEL S. .....145 E. 54th St., OALR
DAVIDSON, MORRIS .....210 W. 70th St., Op.
DAVIS, A. EDWARD .....40 E. 61st St., Op.
DAVIS, G. E. .....1060 Fifth Ave., OALR
DENISON, WARD C. .....20 E. 53rd St., ALR
DEPPING, CHAS. WM. .....17 W. 54th St., ALR
DIXON, G. S. .....1150 Fifth Ave., R
DLUGASCH, LOUIS .....110 E. 36th St., ALR
DOHERTY, W. B. .....150 W. 55th St., Op.
DUNNINGTON, J. H. .....30 W. 59th St., Op.
ELLIS, Z. H. .....58 E. 65th St., Op.
FERNLUND, GEORGE BYRON....8555 118th St., Richmond Hill, OALR
FOWLER, ROBERT HENRY.....153 E. 62nd St., ALR
FOWLKES, JOHN WINSTON .....30 W. 59th St., ALR
FRANKEL, EDWARD, JR. .....152 W. 58th St., OAL

GATEWOOD, W. L. .....330 Park Ave., ALR
GIORDANO, A. J. .....749 E. 183rd St., ALR
GOLDMAN, A. MILTON .....121 E. 60th St., Op.
GOLDMAN, H. G. .....57 W. 58th St., ALR
GOLDSTEIN, ISADORE .....1095 Park Ave., Op.
GOTTLIEB, MARK J. .....2 W. 87th St., ALR
GRESSER, EDWARD BELLAMY .....39 Fifth Ave., Op.
GRUSHLOW, ISRAEL .....271 Central Park, W., ALR
HALLETT, G. DEWAYNE .....274 W. 86th St., Op.
HANCOCK, J. C. .....2 Rector St., Op.
HARDY, LEGRAND H. .....30 E. 40th St., Op.
HARRIS, THOMAS JEFFERSON .....104 E. 40th St., ALR
HARTSHORNE, ISAAC .....30 W. 59th St., Op.
HASKIN, W. H. .....57 W. 57th St., ALR
HAYS, HAROLD M. .....64 E. 58th St., ALR
HETRICK, L. E. .....40 E. 61st St., ALR
HORN, WALTER L. .....1040 Park Ave., ALR
HURD, L. M. .....39 E. 50th St., ALR
HUGHES, WENDEL LOCHEAD .....20 E. 53rd St., Op.
HUNT, WESTLEY MARSHALL .....33 E. 68th St., ALR
HUTCHINSON, A. T. .....30 W. 59th St., ALR
IMPERATORI, C. J. .....108 E. 38th St., ALR
INGERMAN, S. M. .....418 Central Park, W., OALR
JACOBS, S. M. .....1018 E. 163rd St., OALR
JARECKY, HERMAN .....168 W. 86th St., OALR
JEROME, WM. Z. .....436 Fort Washington Ave., ALR
JOHNSON, THOMAS HOLLAND .....30 W. 59th St., Op.
JONES, MARVIN F. .....121 E. 60th St., ALR
JOSEPHSON, EMANUEL M. .....993 Park Ave., ALR
JUDD, H. B. .....40 E. 61st St., ALR
KELLY, JOSEPH DOMINIC .....140 E. 54th St., OALR
KEND, LEE .....400 West End Ave., ALR
KEY, BEN WITT .....100 W. 59th St., Op.
KING, JAMES J. .....133 E. 58th St., ALR
KIRBY, D. B. .....780 Park Ave., Op.
KNAPP, ARNOLD .....10 E. 54th St., Op.
KNIGHTON, W. S. .....40 E. 61st St., Op.
KOPETZKY, S. J. .....51 W. 73rd St., ALR
KRUG, ERNEST F. .....988 Fifth Ave., Op.
LAMBERT, ROBERT .....7 E. 85th St., Op.
LA VIGNE, A. A. .....471 Park Ave., OALR
LEVINE, JOSEPH .....113 E. 61st St., Op.
LEVY, HARRY H. .....State Tower Bldg., Op.
LUBMAN, MAX .....17 E. 96th St., ALR
MCAULIFFE, GERVAIS W. .....110 W. 55th St., ALR
MCCOY, JOHN .....730 Fifth Ave., ALR
MCDANNALD, C. E. .....100 W. 59th St., Op.
MCDONALD, DENNIS J. .....17 E. 38th St., ALR
MCKEOWN, HUGH S. .....30 W. 59th St., Op.
MAY, CHAS. H. .....698 Madison Ave., Op.

MEEK, R. E. ..... 30 E. 40th St., Op.
MEIERHOF, E. L. (Retired) ..... 253 W. 72nd St., OALR
MILLER, JOSEPH WILLIAM ..... 305 E. 17th St., ALR
MINSKY, HENRY ..... 1 W. 86th St., Op.
MITTENDORF, A. D. ..... 339 Park Ave., Op.
MITTENDORF, W. KEITH ..... 115 E. 53rd St., Op.
MORRISON, WM. WALLACE ..... 39 E. 50th St., ALR
MOYLE, EUGENE H. ..... 1088 Park Ave., ALR
MYERSON, M. C. ..... 12 E. 86th St., ALR
NISSELSON, MAX ..... 1018 E. 163rd St., ALR
NORTHCOTT, THOMAS A. ..... 17 E. 38th St., Op.
OAST, SAMUEL P., JR. ..... 30 E. 40th St., Op.
PALMER, ARTHUR ..... 667 Madison Ave., ALR
PETERSON, R. A. ..... 150 Fifth Ave., OALR
REESE, ALGERNON B. ..... 73 E. 71st St., Op.
ROSENBAUM, MORRIS ..... 61 W. 88th St., Op.
ROSENBLUTH, MICHAEL ..... 1018 E. 163rd St., ALR
SAMUELS, BERNARD ..... 57 W. 57th St., Op.
SCAL, JOSEPH COLEMAN ..... 44 W. 74th St., ALR
SCHEER, HENRY M. ..... 522 West End Ave., ALR
SCHILLER, A. N. ..... 101 Central Park West, ALR
SCHLIVEK, KAUFMAN ..... 1016 Fifth Ave., Op.
SCHMIDT, OTTO V. M. ..... 111 E. 61st St., ALR
SCHOENBERG, MARK J. ..... 1160 Park Ave., Op.
SCHWARTZ, A. A. ..... 941 Park Ave., ALR
SETTEL, NATHAN ..... 10 W. 82nd St., ALR
SEWARD, J. A. ..... 121 E. 60th St., ALR
SITCHEVSKA, OLGA L. ..... 160 W. 87th St., Op.
SMITH, JAS. M. ..... 123 E. 53rd St., ALR
SOMBERG, J. S. ..... 470 Park Ave., Op.
STARK, JESSE B. ..... 45 Park Ave., Op.
STRATSMA, CLARENCE R. ..... 899 Park Ave., ALR
STURGES, LEIGH F. ..... 121 E. 60th St., ALR
TUSAK, E. A. ..... 115 E. 90th St., Op.
WACHSBERGER, ALFRED ..... 51 W. 73rd St., ALR
WEEKS, WEBB WILLIAM ..... 20 E. 53rd St., Op.
WEXLER, DAVID ..... 55 E. 86th St., Op.
WHEELER, I. M. ..... 635 W. 165th St., Op.
WIENER, ALFRED ..... 550 Park Ave., OALR
WINCOR, HENRY G. ..... 2021 Grand Concourse, Op.
YANKAUER, SIDNEY ..... 121 E. 60th St., ALR

NIAGARA FALLS

PRICE, NORMAN W. ..... 445 Third St., OALR
ROOKER, A. M. ..... 203 Gluck Bldg., ALR

NYACK

TOMS, S. W. S. ..... 120 S. Broadway, OALR

ONEIDA

CROCKETT, R. L. ..... 302 Main St., Op.

PLATTSBURG

SCHNEIDER, ANTON S. .....56 Court St., Op.A

POUGHKEEPSIE

KRIEGER, WM. A. .....103 Hooker Ave., OALR

ROCHESTER

BARBER, F. .....75 S. Fitzhugh St., Op.
BARLOW, ROY A. .....Medical Arts Bldg., 277 Alexander St., ALR
CARROLL, GEORGE G. .....5 North Goodman St., OALR
CLARK, L. H. .....337 Monroe Ave., OALR
GIPNER, J. F. .....53 S. Fitzhugh St., Op.
JONES, L. W. .....16 N. Goodman St., OALR
KENNEDY, ELDRED WESTON .....Gas and Electric Bldg., Op.
LERNER, MACY LEVI .....332 Park Ave., Op.
MCDOWELL, N. D. .....275 Alexander St., OALR
MORRIS, A. G. .....277 Alexander St., ALR
NASH, C. STEWART .....277 Alexander St., ALR
SHAPERO, I. M. .....289 Alexander St., OALR
SNELL, ALBERT C. .....53 S. Fitzhugh St., Op.

SYRACUSE

BRITTEN, G. S. .....Medical Arts Bldg., ALR
BROWN, MORTIMER G. .....713 E. Genesee, ALR
BRUST, H. O. .....820 S. Crouse Ave., ALR
CAVE, LINUS S. .....221 Physicians Bldg., ALR
FOWLER, S. R. .....University Block, ALR
GILLETTE, D. F. .....State Tower Bldg., Op.
HALL, EDWIN P. .....State Tower Bldg., ALR
HOOPLE, GORDON D. .....Medical Arts Bldg., ALR
JOY, HAROLD HENRY .....504 State Tower Bldg., Op.
KAUFFMAN, A. B. .....Chimes Bldg., ALR
KLINE, H. G. .....State Tower Bldg., ALR
MARLOW, SEARLE B. .....1004 State Tower Bldg., Op.
MOORE, ROY S. .....Medical Arts Bldg., ALR
RUBENSTEIN, A. H. .....Medical Arts Bldg., ALR
WEBSTER, F. R. .....720 State Tower Bldg., Op.

TROY

SULZMAN, FRANK MALCOLM .....1831 Fifth Ave., OALR

UTICA

BEATTIE, WILLIAM H. .....258 Genesee St., OALR
VALONE, FRANK H. .....258 Genesee St., ALR

WATERTOWN

ATKINSON, WALTER S. .....168 Sterling St., Op.
HENDERSON, L. E. .....168 Sterling St., Op.
MCASKILL, JAS. E. .....Woolworth Bldg., ALR

WHITE PLAINS

MORIARTY, RICHARD W. ..... Medical Center, OALR

WOODHAVEN

FROTHINGHAM, EDWARD ..... 8644 94th St., OALR

YONKERS

WOLCOTT, CHAS. C. ..... 291 N. Broadway, ALR

NORTH CAROLINA

ASHEVILLE

GREENE, JOSEPH BERRY ..... Haywood Bldg., ALR
SWANN, CECIL C. ..... 101 Haywood St., ALR

CHARLOTTE

MATHESON, J. P. ..... 106 W. 7th St., ALR
PEELER, C. N. ..... 106 W. 7th St., OALR
SLOAN, HENRY L. ..... 106 W. 7th St., Op.
WHISNANT, A. M. ..... 517 Professional Bldg., OALR

GREENSBORO

REAVES, CHAS. R. ..... Jefferson Standard Bldg., OALR
REAVES, W. P. ..... Reaves Infirmary, 117 W. Sycamore St., OALR

KINSTON

PEERY, VANCE PRICE ..... ALR

MOREHEAD CITY

JOHNSON, OTIS H. ..... OALR

NEW BERN

DANIELS, R. L. ..... Box 685, OALR

RALEIGH

HICKS, VONNIE MONROE ..... Professional Bldg., Op.
WRIGHT, J. B. ..... Professional Bldg., ALR

WILMINGTON

MURPHY, J. G. ..... North Carolina Bank Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W. ..... 210 O'Hanlon Bldg., OALR

NORTH DAKOTA

BISMARCK

BUCKINGHAM, TRACY W. ..... Cowan Bldg., ALR
CONSTANS, GEORGE MAURICE ..... Clinic Bdlg., Op.

FARGO

RINDLAUB, ELIZABETH P. .....de Lendrecie Bldg., OALR
RINDLAUB, JOHN H. .....de Lendrecie Bldg., OALR

MINOT

MCCANNEL, A. D. .....15A Central Ave. W., OALR

OHIO

AKRON

ARBUCKLE, DAVID S. .....513 W. Market St., ALR
BROWN, L. E. .....Second Nat'l Bank Bldg., OALR
MOORE, T. K. .....Second Nat'l Bank Bldg., OALR

ALLIANCE

KING, GEORGE L., JR. .....537 Market St., Op.
KING, G. L., SR. .....537 E. Market St., OALR

BELLEFONTAINE

BUTLER, ROBERT H. .....112 W. Columbus St., OALR
HARBERT, JOHN P. .....135 N. Main St., OALR

CANTON

HOFFMANN, JOHN N. .....309 Brant Bldg., ALR
SCHILD, E. H. .....Brant Bldg., Op.
TOOT, JOSEPH F. .....923 First Nat'l Bank Bldg., Op.
WEAVER, HOMER V. .....228 Cleveland Ave., N.W., ALR

CINCINNATI

ABRAHAMSON, IRA A. .....605 Traction Bldg., Op.
ASBURY, MARY K. .....3549 Holly Lane, Op.
BEYER, A. G. .....Doctors Bldg., OALR
BROWN, ALBERT LOUIS .....Doctors Bldg., Op.
BRYANT, BEN L. .....19 Garfield Place, ALR
FISCHBACH, VICTOR W. .....Union Central Bldg., ALR
GOODYEAR, H. M. .....Doctors Bldg., ALR
HANS, C. L. .....Provident Bank Bldg., OALR
HARRIS, W. C. .....Groton Bldg., ALR
HENDICKS, LOUIS J. .....210 W. 12th St., Op.
HOFLING, CHARLES A. .....707 Race St., Op.
HOWARD, ROBERT E. .....Union Central Bldg., ALR
IGLAUER, SAMUEL .....707 Race St., ALR
JONES, C. C. .....656 Doctors Bldg., ALR
KING, CLARENCE .....Union Central Bldg., Op.
KING, ED. .....1002 Carew Tower, ALR
LYLE, DONALD J. .....Carew Tower, OALR
MITHOEFER, W. .....Doctors Bldg., ALR
PHINNEY, F. D. .....22 W. 7th St., OALR
RAY, VICTOR .....Carew Tower, Op.

RAY, VICTOR, JR. ..... Carew Tower, Op.
REID, H. W. ..... 19 Garfield Place, Op.
SELTZ, SAMUEL ..... Provident Bank Bldg., ALR
SIEGEL, FRANCIS X. ..... 901 Union Central Life Bldg., OALR
STANBERRY, HENRY ..... 566 Doctors Bldg., Op.
STEWART, T. M. ..... 901 Union Trust Bldg., OALR
STITT, H. L. ..... 656 Doctors Bldg., ALR
STOLL, LUDWIG ..... 19 West 7th St., OALR
TANGEMAN, H. F. ..... 606 Union Central Life Bldg., ALR
THOMPSON, E. H. ..... 8 W. 9th St., OALR
URNER, M. H. ..... 2600 Union Central Bldg., OALR
VAIL, D. T., JR. ..... Carew Tower, 441 Vine St., Op.
VAIL, H. H., SR. ..... Carew Tower, 441 Vine St., ALR

CLEVELAND

ABBOTT, W. J. ..... Carnegie Medical Bldg., OALR
BRUNER, ABRAM BRENEMAN ..... 1214 Guardian Bldg., OALR
BRUNER, WM. E. ..... Guardian Bldg., Op.
BURKE, T. A. ..... Rose Bldg., OALR
CHAMBERLAIN, WEBB PARKS ..... 7405 Detroit Ave., Op.
DIXON, FRED WILLIS ..... 1029 Rose Bldg., ALR
FORSYTHE, S. T. ..... Rose Bldg., OALR
KENDALL, M. RAYMOND ..... Hanna Bldg., ALR
LARGE, S. H. ..... Hanna Bldg., ALR
LENKER, JOHN N. ..... Osborne Bldg., ALR
MCDONALD, C. L. ..... 964 Rose Bldg., OALR
METZ, ROY B. ..... Hanna Bldg., Op.
METZENBAUM, MYRON ..... Carnegie Medical Bldg., ALR
MITCHELL, HORACE E. ..... 15701 Detroit Ave., ALR
MONSON, S. H. ..... 1322 B. F. Keith Bldg., OALR
MOORE, PAUL G. ..... Medical Arts Bldg., Op.
MOTTO, MICHAEL PAUL ..... Rose Bldg., Op.
MULLIN, WM. V. .... Cleveland Clinic, Euclid Ave. at 93rd St., ALR
MUSSUN, W. G. ..... Medical Arts Bldg., ALR
NELSON, CHAS. F. ..... 606 Schofield Bldg., OALR
PITKIN, C. E. ..... Carnegie Medical Bldg., ALR
PRENDERGAST, D. A. ..... Medical Arts Bldg., OALR
PUTNAM, F. I. ..... 1401 Prospect Ave., OALR
QUITTNER, S. S. ..... 10515 Carnegie Ave., OALR
RIEGER, W. H. ..... Hanna Bldg., ALR
ROSENBERGER, HARRY CLIFFORD ..... Rose Bldg., ALR
RUEDEMANN, ALBERT DARWIN ..... Cleveland Clinic, Op.
RUST, E. G. ..... Hanna Bldg., Op.
SHACKLETON, W. E. ..... 2323 Prospect Ave., Op.
SHIRAS, HOWARD H. ..... 624 Guardian Bldg., Op.
SHIREY, O. M. ..... Osborn Bldg., ALR
SIMONDS, O. F. ..... Hanna Bldg., Op.
STOTTER, ARTHUR L. ..... 1148 Euclid Ave., OALR
TUCKERMANN, W. C. ..... 733 Osborn Bldg., Op.

TUCKERMANN, W. H. .....733 Osborn Bldg., OALR
WAUGH, J. M. .....Cleveland Clinic, Euclid Ave. at 93rd St., OALR
WEIDLEIN, IVAN F. .....10515 Carnegie Ave., ALR

COLUMBUS

BEATTY, H. G. .....681 E. Broad St., ALR
BROWN, JOHN E. .....370 E. Town St., OALR
CLARK, I. G. .....188 E. State St., OALR
DAVIES, MORGAN C. .....269 E. State St., Op.
DAVIS, WM. C. .....327 E. State St., Op.
FROST, ALBERT DALBEY .....681 E. Broad St., Op.
HARRIS, ED. WM. .....681 E. Broad St., ALR
HAUER, A. M. .....327 E. State St., ALR
HELFRICH, ED. D. .....269 E. State St., ALR
JOHNSON, DAVID M. .....327 E. State St., ALR
MEANS, R. G. .....40 S. Third St., ALR
PERRY, CLAUDE S. .....40 S. Third St., Op.
POSTLE, C. D. .....240 E. State St., ALR
PROUT, A. W. .....380 E. Town St., OALR
SANOR, DANIEL G., JR. .....206 E. State St., ALR
SILBERNAGEL, C. E. .....240 E. State St., OALR
STOUTENBOROUGH, WM. A. .....21 E. State St., OALR
SULZER, G. A. .....327 E. State St., OALR
THOMAS, FRANCIS W. .....327 E. State St., OALR
TIMBERMAN, ANDREW .....Beggs Bldg., OALR

DAYTON

BINKLEY, ROY S. .....Fidelity Bldg., OALR
BLACKBURN, W. J. .....663 Reibold Bldg., OALR
CULLER, ARTHUR M. .....Fidelity Medical Bldg., Op.
DUTROW, H. V. .....1040 Fidelity Medical Bldg., OALR
FARMER, A. G. .....Fidelity Medical Bldg., OALR
FOUTS, JOHN D. .....Fidelity Medical Bldg., OALR
HARRIS, H. B. .....Fidelity Medical Bldg., OALR
KILBOURNE, P. H. .....Fidelity Medical Bldg., OALR
MILLETTE, J. W. .....Reibold Bldg., OALR

IRONTON

DUNN, O. B. .....Fourth and Railroad Sts., OALR

LAKEWOOD

MERICA, F. W. .....14805 Detroit Ave., ALR

LIMA

STUEBER, F. G. .....209 W. North St., OALR
STUEBER, PAUL J. .....209 W. North St., OALR

LORAIN

MONOSMITH, O. B. .....O. P. S. Bldg., OALR

MANSFIELD

GARBER, J. M. ..... 14 N. Mulberry St., OALR
WISE, RALPH C. ..... News Bldg., OALR

MARIETTA

SAUER, W. W. ..... 231 Third St., OALR

MARION

BRICKLEY, DANIEL W. ..... Uhler-Phillips Bldg., OALR

MASSILLON

SCOTT, MERLE E. ..... First Nat'l Bank Bldg., OALR

MIDDLETOWN

WILLIAMS, WALTON H. ..... Castell Bldg., OALR

NEWARK

HATCH, C. B. ..... 3 W. Church St., OALR

SPRINGFIELD

EASTON, J. C. ..... First Nat'l Bank Bldg., OALR
HARTLEY, F. A. ..... 302 E. High St., OALR
HOGUE, D. W. ..... 34 W. High St., OALR
MINOR, C. L. ..... First Nat'l Bank Bldg., OALR

STEUBENVILLE

GOURLEY, G. F. ..... Sinclair Bldg., OALR

TIFFIN

PORTER, E. H. ..... 85 Madison St., OALR

TOLEDO

ALDERDYCE, WM. W. ..... 513 Madison Ave., OALR
DENMAN, IRA O. ..... 330 Richardson Bldg., OALR
KING, CHAS. R. ..... Spitzer Bldg., OALR
LUKENS, CHARLES ..... 316 Michigan St., OALR
LUKENS, JOHN A. ..... 316 Michigan St., ALR
NIPPE, LEONARD ..... 316 Michigan St., OALR
SNYDER, W. H. ..... 2001 Colingwood Ave., OALR
STEINFELD, A. L. ..... 125 Fifteenth St., OALR
WINGER, IRA B. ..... 225 Michigan St., OALR

WARREN

STIMSON, GEORGE W. ..... Packard Bldg., OALR

XENIA

MADDEN, REED ..... Allen Bldg., OALR
SHIELDS, LAWRENCE ..... Allen Bldg., ALR

YOUNGSTOWN

BEARD, HAROLD JOLLY .....510 Dollar Bank Bldg., OALR
GOLDCAMP, ED. C. .....Dollar Bank Bldg., ALR
HARTZELL, SOL. M. .....260 Tod Lane, OALR

ZANESVILLE

CULBERTSON, L. R. .....Masonic Temple, OALR

OKLAHOMA

ENID

PIPER, ARTHUR S. .....610 S. Monroe, OALR

FORT SILL

DEWEY, MAJ. C. H. .....M. C., U. S. Army, Artillery School, OALR

GUTHRIE

BARKER, CHAS. B. .....224½ W. Okla. Ave., OALR

McALESTER

DAVIS, J. E. .....Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M. .....Barnes Bldg., OALR

OKLAHOMA CITY

FERGUSON, E. S. .....902 Medical Arts Bldg., OALR
GUTHRIE, A. L. .....Osler Bldg., OALR
MACDONALD, J. C. .....301 W. 12th St., OALR
McHENRY, L. C. .....Medical Arts Bldg., ALR

TULSA

BRASWELL, JAMES C. .....Medical Arts Bldg., OALR
COOK, W. ALBERT .....Medical Arts Bldg., OALR
HARALSON, CHARLES HERNDON .....Medical Arts Bldg., Op.
HUBER, WALTER A. .....Medical Arts Bldg., Op.
ROTH, ALBERT W. .....Medical Arts Bldg., OALR
SMITH, RURIC N. .....Medical Arts Bldg., ALR

OREGON

BAKER

GORDON, GLENN G. .....First Nat'l Bank Bldg., ALR

EUGENE

NORTON, ARTHUR H. .....1162 Williamette, Op.

LA GRANDE

BOUVY, L. B. .....Foley Bldg., OALR

PORTLAND

AINSLIE, GEORGE ..... Selling Bldg., OALR
BOUVY, HARRY M. ..... 385½ Washington, OALR
BRUERE, GUSTAVE E. ..... Medical-Dental Bldg., OALR
CARRUTH, HOWARD ERNEST ..... Medical Arts Bldg., OALR
DAVIS, RALPH F. ..... Mayer Bldg., OALR
FENTON, RALPH A. ..... 806 Medical Arts Bldg., OALR
HENTON, G. E. ..... Morgan Bldg., OALR
JOHNSTON, WILSON ..... 611 Lovejoy St., OALR
KARKEET, ROBERT B. ..... Medical-Dental Bldg., OALR
KIEHLE, F. A. ..... 406 Taylor St., OALR
KISTNER, F. B. ..... 600 Mayer Bldg., OALR
LUCAS, HAROLD R. ..... Medical-Dental Bldg., OALR
LUPTON, IRVING MARTIN ..... Medical Arts Bldg., OALR
PETHERAM, C. C. ..... Medical Arts Bldg., ALR
TAYLOR, ED. M. ..... Medical Arts Bldg., Op.
WEEKS, JOHN E. ..... 260 Edgecliff Road (Retired), Op.

SALEM

BAUM, WM. W. ..... Guardian Bldg., ALR
CLEMENT, L. O. ..... Guardian Bldg., OALR

PENNSYLVANIA

ALLENTOWN

UHLER, S. MANN ..... 104 N. Eighth St., ALR

ALTOONA

BUZZARD, JOSIAH F. ..... 1110 Thirteenth Ave., Op.
GLOVER, S. P. ..... 1118 Twelfth Ave., OALR

BEAVER

CRUMRINE, NORMAN ROSS ..... 595 Third St., OALR
SCROGGS, J. J. ..... 218 E. Third St., Op. A

BETHLEHEM

CHASE, W. D. ..... 230 E. Broad St., ALR

BUTLER

DOANE, L. L. ..... 357 N. Main St., OALR
HAZLETT, L. R. ..... Savings and Trust Bldg., OALR

CHESTER

CROSS, G. H. ..... 525 Welsh St., Op.
ORR, ADRIAN V. B. ..... 525 Welsh St., ALR

CONNELLSVILLE

HARRISON, FRED H. ..... 308 First Nat'l Bank Bldg., OALR

DEVON

O'Reilly, Chas. A. ..... ALR

DU BOIS

Beals, C. Wearne ..... 41 N. Brady St., ALR

EASTON

Bloom, C. Hugh ..... 65 N. Third St., ALR

EMLENTON

Ferman, John W. ..... OALR

ERIE

Delaney, Jas. H. ..... 138 W. 9th St., Op.
Dennis, Edward P. ..... 309 W. 10th St., OALR
Fust, John H. E. ..... 221 W. 9th St., OALR
Jackson, J. D. ..... 232 W. 8th St., OALR
Russell, J. A. Merle ..... 813 Sassafras St., OALR
Schlindwein, G. Wm. ..... 138 W. 9th St., OALR
Shreve, Owen M. ..... 162 W. 8th St., Op.

FRANKLIN

Jobson, G. B. ..... West Park and Liberty St., OALR

GREENSBURG

Fairing, John W. ..... Tribune Review Bldg., OALR
McKee, Claud W. ..... Coulter Bldg., OALR

HARRISBURG

Dailey, Gilbert L. ..... 618 N. 3rd St., OALR
Farnsler, H. Hershey ..... 1438 Market St., OALR
Gracey, George F. ..... 209 State St., OALR

HAZLETON

Reiche, O. C. ..... Markle Bank Bldg., Op.

JOHNSTOWN

Barker, O. G. A. ..... 804 Johnstown Trust Bldg., Op.
Griffith, Harold M. ..... 804 Johnstown Trust Bldg., Op.
Harris, C. M. ..... 604 Johnstown Trust Bldg., OALR
Rush, C. C. ..... 907 U. S. Nat'l Bank Bldg., OALR

LEBANON

Reese, Monroe D. ..... 123 N. 8th St., OALR

MIDDLETOWN

George, Henry W. ..... 19 N. Union St., Op.

NEW KENSINGTON

McCONNELL, T. E. .....Logan Trust Bldg., OALR
STEIM, J. M. .....First Nat'l Bank Bldg., Op.

NORRISTOWN

BEIDEMAN, JOSEPH ELLSWORTH....Montgomery Trust Arcade, Op.
PRICE, JOHN BEADLE .....824 De Kalb St., ALR

PHILADELPHIA

ADLER, FRANCIS HEED .....313 S. 17th St., Op.
ALEXANDER, GEORGE J. .....1930 Chestnut St., ALR
ANNON, W. T. .....235 S. 15th St., ALR
APPLEMAN, L. F. .....308 S. 16th St., Op.
BABBITT, JAMES A. .....1912 Spruce St., ALR
BAER, BENJAMIN F., JR. .....1927 Spruce St., Op.
BALDWIN, KATE W. .....1117 Spruce St., ALR
BALENTINE, P. L. .....1524 Chestnut St., Op.
BLAIR, MORTIMER W. .....N.E. Cor. 20th and Chestnut St., Op.
BOEHRINGER, H. WINFIELD .....700 Central Medical Bldg., OALR
BRICKER, SACKS .....3701 N. Broad St., ALR
BROWN, SAMUEL HORTON .....1930 Chestnut St., Op.
BRUMM, SETH A. .....Medical Arts Bldg., OALR
BURNS, L. J. .....1930 Chestnut St., ALR
BUTT, MIRIAM M. .....2045 Chestnut St., Op.
CARPENTER, C. .....1930 Chestnut St., Op.
CLERF, LOUIS HENRY .....128 S. 10th St., ALR
COATES, GEORGE M. .....1721 Pine St., ALR
COHEN, HERMAN B. .....The Lenox, Spruce and 13th Sts., ALR
COHEN, SAMUEL .....2009 Pine St., ALR
COWAN, ALFRED .....1930 Chestnut St., Op.
CREIGHTON, WM. J. .....258 S. 18th St., Op.
DAVIS, J. L. .....135 S. 18th St., ALR
DAVIS, T. CARROLL .....3128 N. Broad St., ALR
DEICHLER, LYN W. .....1930 Chestnut St., OALR
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ERSNER, MATTHEW S. .....1915 Spruce St., ALR
EVES, C. C. .....1910 Spruce St., ALR
FEWELL, A. G. .....1924 Pine St., Op.
FRY, W. E. .....1912 Spruce St., Op.
GLEASON, E. B. .....2112 Pine St., ALR
GODDARD, H. M. .....1531 Spruce St., ALR
GOUTERMAN, JOSEPH I. .....1930 Chestnut St., Op.
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GRIMES, CHAS. H. .....3546 N. Broad St., ALR
GRISCOM, J. MILTON .....2213 Walnut St., Op.
HAWMAN, ERLE G. .....4051 N. Broad St., Op.
HEED, C. R. .....1205 Spruce St., Op.
HEWSON, WM. .....6013 Greene St., Germantown, ALR

HIRST, OTTO C. .....1930 Chestnut St., ALR
HOLLOWAY, THOMAS B. .....1819 Chestnut St., Op.
HUNTER, R. J. .....2011 Chestnut St., ALR
HUSIK, D. N. .....1930 Chestnut St., ALR
JACKSON, CHEVALIER .....3432 N. Broad St., L
KAUFMAN, A. SPENCER .....1923 Spruce St., ALR
KEELER, J. C. .....Medical Arts Bldg., ALR
KRAUSS, FREDERICK .....1930 Chestnut St., OALR
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LOFTUS, JOHN EDWARD .....1930 Chestnut St., ALR
LUKENS, ROBERT McD. .....1923 Spruce St., ALR
LUONGO, R. A. .....1713 Pine St., ALR
MCANDREWS, LEO F. .....1930 Chestnut St., Op.
MACKENZIE, ALICE V. .....1724 Spruce St., OALR
MACKENZIE, GEORGE W. .....1724 Spruce St., OALR
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MOORE, GEORGE H. .....1737 Chestnut St., OALR
MOORE, WILLIAM FREDERICK .....255 S. 16th St., ALR
OLSHO, S. L. .....235 S. 15th St., OALR
PERSKY, ABRAM H. .....1922 Spruce St., ALR
PETER, L. C. .....1930 Chestnut St., Op.
PONTIUS, P. J. .....1831 Chestnut St., Op.
REESE, WARREN S. .....230 S. 21st St., Op.
RIDPATH, ROBERT F. .....N.E. Cor. 18th and Chestnut Sts., LR
ROMMEL, JOHN C. .....3701 N. Broad St., Op.
RUDOLPHY, JAY BESSON .....1924 Pine St., Op.
SARTAIN, PAUL J. .....2006 Walnut St., Op.
SCARLETT, HUNTER W. .....230 S. 21st St., Op.
SCHWEINITZ, GEORGE E. DE .....1705 Walnut St., Op.
SENER, WALTER J. .....Physicians Bldg., Op.
SHANNON, C. E. G. .....1930 Chestnut St., Op.
SHEMELEY, WM. G., JR. .....1724 Spruce St., OALR
SHUSTER, BENJAMIN HARRISON .....1930 Chestnut St., ALR
SKILLERN, S. R., JR. .....1734 Pine St., ALR
SLOANE, HENRY O. .....1717 Pine St., Op.
SLOTKIN, HERMAN B. .....1819 Pine St., ALR
SPAETH, EDMUND B. .....1930 Chestnut St., Op.
SPENCER, W. H. .....1830 S. Rittenhouse Sq., ALR
STILES, CHAS. M. .....1130 Spruce St., Op.
STOUT, PHILIP S. .....269 S. 19th St., ALR
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CONWAY, JOHN M. .....5151 Jenkins Arcade, ALR
COOPER, ROY C. .....510 S. Aiken Ave., ALR
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DAY, KENNETH M. .....121 University Pl., ALR
DAY, EWING W. .....121 University Pl., OALR
FISCHER, N. ARTHUR .....623 Union Trust Bldg., ALR
GROSS, JULIUS E. .....8030 Jenkins Arcade Bldg., ALR
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HAGEMANN, JOHN A. .....Highland Bldg., ALR
HAMPSEY, A. R. .....806 May Bldg., ALR
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MCCREADY, J. HOMER .....121 University Pl., ALR
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MARKEL, J. CLYDE .....Westinghouse Bldg., Op.
MARSHALL, WATSON .....121 University Pl., ALR
MEANOR, WM. C. .....619 Westinghouse Bldg., Op.
PATTERSON, ELLEN J. .....121 University Pl., ALR
PLUMER, JOHN S. .....121 University Pl., Op.
SEEGMAN, SIMON .....Medical Bldg., ALR
SHUMAN, G. H. .....Park Bldg., Op.
SIMPSON, JOHN REID .....825 Highland Bldg., ALR
SMITH, STANLEY S. .....121 University Pl., Op.
STIEREN, E. .....626 Union Trust Bldg., Op.
STURM, S. A. .....7036 Jenkins Arcade Bldg., Op.
TAYLOR, M. C. .....May Bldg., Op.
THORPE, H. E. .....8075 Jenkins Arcade Bldg., Op.
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TURNER, H. H. .....902 Professional Bldg., Op.
WEISSER, E. A. .....806 May Bldg., Op.
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POSEY, WM. C. .....Op.

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RHODE, SOLON L. .....238 N. 6th St., OALR
STOCKLER, J. A. .....332 N. 5th St., OALR

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CARR, GEORGE W. .....54 S. Franklin St., OALR
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CAPRON, F. P. .....118 Angell St., OALR
HARVEY, N. D. .....112 Waterman St., OALR
LEECH, J. W. .....369 Broad St., OALR
MESSINGER, H. C. .....210 Angell St., Op.
MUNCY, WM. M. .....162 Angell St., OALR
PORTER, L. B. .....199 Thayer St., OALR
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EZELL, H. C. .....228 Doctors Bldg., OALR
HASTY, F. E. .....Medical Arts Bldg., ALR
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ORR, EUGENE .....Doctors Bldg., ALR
SULLIVAN, ROBT. ED. .....Doctors Bldg., Op.
WARNER, ROBERT JAY .....Doctors Bldg., Op.
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TEXAS

AMARILLO

CRUME, JOHN JAS. .....320 Amarillo Bldg., ALR
DUNCAN, ROBERT AUSTIN .....339 Amarillo Bldg., ALR
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AUSTIN

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SCHUSTER, S. A. .....First Nat'l Bank Bldg., ALR
VANDEVERE, WM. E. .....Bassett Tower, ALR

FORT WORTH

GOODMAN, THOMAS L. .....Medical Arts Bldg., ALR
GOUGH, ROY H. .....Medical Arts Bldg., OALR
HOWARD, EDWARD L. .....405 Medical Arts Bldg., OALR
JACKSON, ATRAS E. .....Medical Arts Bldg., Op.
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GILL, WM. DAVIS .....323 Medical Arts Bldg., OALR
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